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Cardiac ultrasound in cardiovascular emergency and critical care: a clinical consensus statement of the European Association of Cardiovascular Imaging, the Acute CardioVascular Care Association of the European Society of Cardiology, and the European Association of Cardiothoracic Anaesthesia and Intensive Care

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Introduction

Cardiovasculardiseases (CVDs) remain a leading cause of morbidity and mortality worldwide.[1] Acute cardiovascular conditions demand immediate attention and rapid, precise diagnostic approaches.[2] Cardiac ultrasound (CUS) offers real-time, non-invasive visualization of cardiac structures and function and dynamic feedback, supporting rapid, accurate decisions. When integrated with lung[3,4] and organ-specific ultrasound,[5] it becomes a powerful tool—multimodal ultrasound assessment of cardiovascular haemodynamics.

This clinical consensus statement serves as a practical guide for the application of CUS in acute care settings. It represents a collaborative effort by experts from various disciplines, aiming to harmonize practices and enhance the standard of care. For each clinical scenario, the document emphasizes a thorough understanding of the underlying pathophysiology, guiding clinicians towards accurate diagnoses by integrating ultrasound findings into a comprehensive diagnostic framework. Furthermore, it addresses key aspects such as training, governance, and future perspectives to ensure sustainable advancement in the field.

General considerations

CUS is widely accessible, however, the diagnostic yield must be carefully considered according to the device used [e.g. handheld ultrasound device (HUD) vs. high-end echocardiography], the level of training and experience [e.g. Focus CUS (FoCUS) practitioner vs. fully trained echocardiographer], and the acuity and setting [e.g. echocardiography laboratory vs. emergency department/intensive care unit (ICU); *Table 1*]. Frequently used echocardiographic parameters and their pitfalls in cardiovascular emergency and critical care are listed in *Table 2*. Further, correct interpretation requires adequate contextualization with ongoing respiratory/cardiovascular support and inherent patient constraints. The different types of imaging undertaken in the acute setting and the relevant terminology used are shown in *Figure 1*. It is important to note that the type of imaging performed, and its level of complexity will be determined by the clinician's expertise and the specific clinical setting.

In addition, *Table 3* summarizes the appropriateness criteria for the use of CUS in cardiac and cardiac-like emergencies.

Objectives of this clinical consensus statement

The primary objective of this clinical consensus statement is to establish a practical guide for the use of CUS in cardiovascular emergencies and critical care settings. This provides the update of the previous EACVI document which focused on emergency echocardiography,[25] and

Table 1 Definitions and terminology of cardiac ultrasound in cardiovascular emergencies and critical care

Term	Definition	Applications
Emergency echocardiography	Comprehensive echocardiographic examination is performed in an emergency setting in the assessment of patients with unstable CVDs by an operator fully trained in echocardiography, which acquires a well-defined data set.	Full assessment of cardiac morphology and function
FoCUS	A specific type of PoCUS applied to the heart, as an extension of the clinical examination, by an operator not necessarily trained in comprehensive echocardiography, but appropriately trained in FoCUS, usually responsible for decision-making and/or treatment.	Rapid assessment of global left and RV function, estimating volume status, detecting pericardial effusion/tamponade
PoCUS	A goal-oriented, bedside, limited ultrasound examination, extending physical examination performed in any body structure and environment with a predefined limited protocol. Often involves cardiac and non-cardiac assessments (i.e. lung, venous, neurological and abdominal ultrasound). Can be performed on a basic to advanced operator level.	Bedside multi-organ ultrasound assessment to guide clinical bedside decision-making
Targeted (goal-oriented) echocardiography	A targeted echocardiographic examination performed by an operator fully trained in echocardiography.	Attempting to obtain an answer to a specific, often critical and frequently complex clinical dilemma
CCE	CCE involves comprehensive assessment using both standard and advanced echocardiographic techniques to evaluate cardiac function, haemodynamics, assessing preload, afterload, and cardiac output as well as pulmonary and SVR. This requires advanced-level training in CCE.	Monitoring cardiac function, haemodynamics, treatment response in shock, ARDS, guiding the insertion, monitoring, troubleshooting and weaning from MCS devices
fTOE:	This involves inserting an ultrasound probe into the oesophagus to obtain detailed images of the heart by a fully trained echocardiographer. fTOE provides superior image quality, especially in critically ill patients where transthoracic windows are inadequate. This requires comprehensive training on TOE.	It is particularly useful in detecting aortic dissection, endocarditis, for guiding cannulation and troubleshooting from ECMO and MCS, and also useful during cardiopulmonary resuscitation.

FoCUS, focused cardiac ultrasound; PoCUS, point of care ultrasound; CCE, critical care echocardiography; ARDS, acute respiratory distress syndrome; ECMO, extracorporeal membrane oxygenation; fTOE, focused transoesophageal echocardiography.

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Table 2 Echocardiographic parameters and their pitfalls in cardiovascular emergency and critical care

	Parameter	Clinical applications	Pitfalls
LV function and cardiac output	EF	Not validated in critical care settings[6]	Greatly influenced by loading conditions, heart rate, and geometric assumptions[7,8]
	LVOT VTI, stroke volume, cardiac output, and cardiac index	• Quantification of stroke volume and cardiac output • Well correlated to other standard invasive methods of estimation of CO[9] • TTE; determined by LVOT area (obtained by measuring LVOT diameter in parasternal long-axis view in mid-systole then calculating LVOT area multiplied by the LVOT VTI from the apical five-chamber view. • Normal LVOT VTI is in the range of 18–22 cm, and it could be used as a surrogate for SV. • TOE: calculated by obtaining the deep transgastric five-chamber view at 0° or the modified transgastric long-axis view at 120° as both provide fair alignment with the LVOT flow and the PWD beam.	• Incorrect placement of the Doppler sample volume can lead to inaccurate measurements[10] • Variations in LVOT anatomy, such as calcification or stenosis, can affect the accuracy of VTI measurements[11] • Changes in heart rate can affect VTI, making it less reliable in patients with arrhythmias. An average of 10 beats is needed in patients with atrial fibrillation[12] • The Doppler angle should be aligned parallel to blood flow; incorrect angles can lead to underestimation of velocities[13] • Variations in intrathoracic pressure during the respiratory cycle can affect VTI measurements[14] • The experience and skill of the operator significantly impact the accuracy and reproducibility of VTI measurements[15] • Errors in measuring or assuming a constant LVOT diameter can lead to inaccurate calculation of stroke volume and cardiac output, e.g. a large LVOT area can compensate for a reduced VTI leading to adequate SV[16] • Presence of intracardiac shunts can alter flow patterns, leading to incorrect VTI interpretation[17] • Conditions such as hypertrophic cardiomyopathy and dynamic LVOT obstruction can cause overestimation of LVOTI VTI; therefore, it becomes unreliable to assess stroke volume. • Significant AR can lead to increased LV end-diastolic volume, therefore, overestimated LVOT VTI. • Artefacts from improper gain settings, tissue motion, or poor acoustic windows can interfere with accurate Doppler signal acquisition. • Differences in ultrasound machine quality and settings can affect the consistency of measurements.
	E/*e*′	• Estimation of LV diastolic function and filling pressures	• Positive pressure ventilation can alter the intrathoracic pressure, reducing both *E* and *e*′ velocities thereby altering *E*/*e*′ measurement[18] • High heart rates and irregular rhythms can lead to inaccurate *E* and *e*′ wave measurements[19] • Factors like increased intra-abdominal pressure can impact cardiac filling pressures and the *E*/*e*′ ratio[20] • The ratio needs to be interpreted in the context of comprehensive clinical and echocardiographic assessment[14] • Rapid changes in preload or afterload (e.g. fluid resuscitation or vasopressor use) can quickly alter the *E*/*e*′ ratio,[14] LBBB can cause asynchronous contraction and relaxation of the ventricles, affecting *E*/*e*′ accuracy.[21] The presence of prosthetic mitral or aortic valves can interfere with accurate Doppler

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Table 2 Continued

	Parameter	Clinical applications	Pitfalls
			measurements.[22] LV hypertrophy can cause diastolic dysfunction that may not be accurately reflected by *E*/*e*′.[21] Localized wall motion abnormalities can result in inaccurate tissue Doppler velocities, thus affecting *e*′.[19] May be unreliable in patients with severe mitral annular calcification. Limited in patients with significant MR. Changes in fluid status can significantly affect the *E*/*e*′ ratio, making it an unreliable indicator of LV filling pressures.[14] Conditions such as RV pressure overload or septal infarction can affect *e*′ values[21]
Right heart	TAPSE	• Measures longitudinal RV function reflecting intrinsic RV function • Cut-off ≤16 mm	• It only reflects longitudinal function of the free wall. • May be affected by overall heart motion and not solely by RV function[11] • Reduced after cardiac surgery and cardiopulmonary bypass.
	S′ wave (tricuspid lateral annular systolic velocity)	• Tissue Doppler-derived systolic velocity at the lateral tricuspid annulus • Cut-off abnormal <9.5 cm/s	• Influenced by loading conditions and overall heart motion • Requires optimal angle of insonation (<20°)
	RV FAC	• Percentage change in RV area from diastole to systole • Cut-off abnormal <35%	• Poor reproducibility in critically ill • 2D measurement that can be affected by imaging plane and RV geometry
	RV MPI[23,24]	• IVCT + IVRT/RVET • Combines systolic and diastolic time intervals • Cut-off abnormal > 0.43 (PWD), >0.54 (TDI)	• May be influenced by heart rate, preload, and afterload conditions • Complex to calculate accurately • Not validated in the acute setting
	RVEF	• Volumetric measure of RV function • Cut-off abnormal < 45%	• Requires 3D echocardiography for accurate measurement • Operator-dependent and influenced by image quality
	RVEDA	• RV size at end-diastole • Cut-off abnormal > 20 cm^2 (indexed to BSA)	• Can be affected by loading conditions and overall heart size • Measurement variability
	RVESA	• RV size at end-systole • Cut-off abnormal > 10 cm^2 (indexed to BSA)	• Similar pitfalls as RVEDA
	TR velocity	• Assesses RV systolic pressure • Cut-off abnormal >2.8 m/s	• Depends on adequate Doppler signal • May underestimate pressure in severely RV impairment
	RV/LV diameter ratio	• Cut-off abnormal > 0.6	• Influenced by loading conditions and overall heart geometry • Variability in measurement techniques
	IVC size and collapse	• Assesses RV preload • Cut-off IVC size abnormal >2.1 cm with <50% collapse	• Can be influenced by intravascular volume status, mechanical ventilation, intra-abdominal pressure, severe TR, RV failure, biventricular cardiomyopathy, and pericardial constraint
	Septal shift	• Abnormal movement or flattening of the interventricular septum	• Subjective assessment that can be influenced by overall heart motion, LBBB, pacing, and loading conditions
	RA size	• RA enlargement indicates RV dysfunction • Cut-off RA area abnormal >18 cm^2	• Measurement variability and dependence on loading conditions. • Often an indirect measure of chronic RV pressure overload rather than acute dysfunction.

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Table 2 Continued

Parameter	Clinical applications	Pitfalls
RV STE	• RV speckle tracking imaging can be applied to the RV to detect subtle dysfunction that might not be apparent with conventional measures. • RV GLS > −20% is considered abnormal. RV FwLS > −23% is an indicator of RV dysfunction.	• Not yet widely available in emergency and critical care. • It requires an optimum 2D image quality with clear endocardial border, stable heart rate and clean ECG signal for gated studies which is often difficult to achieve in acute settings. • Data are lacking on the value of strain imaging in cardiac critical care patients.

EF, ejection fraction; LVOT, left ventricular outflow tract; VTI, velocity time integral; TTE, transthoracic echocardiography; SV, stroke volume; PWD, pulsed-wave Doppler; LBBB, left bundle branch bock; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; MPI, myocardial performance index; TDI, tissue Doppler imaging; 3D, three-dimensional; RV, right ventricle; RA, right atrium; LV, left ventricle; TR, tricuspid regurgitation; RVEDA, right ventricular end-diastolic area; RVESA, right ventricular end-systolic area; FAC, fractional area change; MPI, myocardial performance index; RVEF, right ventricular ejection fraction; BSA, body surface area; IVC, inferior vena cava; STE, speckle tracking echocardiography; ECG, electrocardiogram.

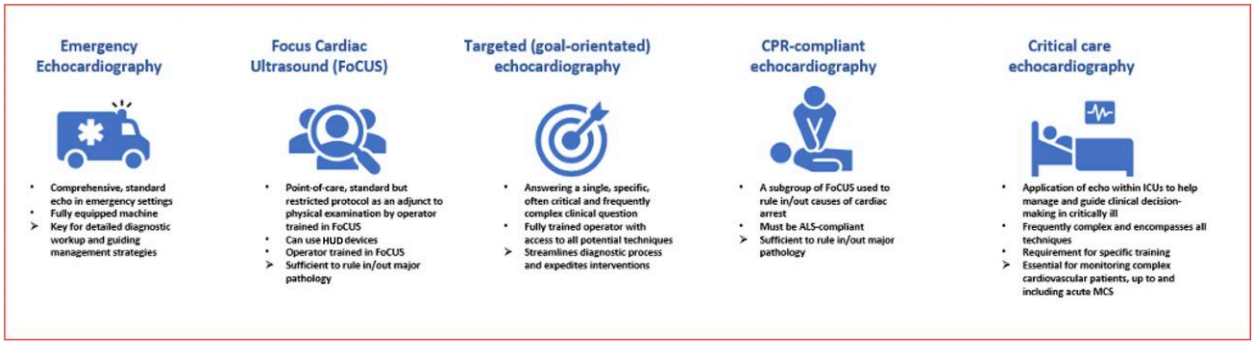
Emergency Echocardiography
Comprehensive, standard echo in emergency settings
Fully equipped machine
Key for detailed diagnostic workup and guiding management strategies
Focus Cardiac Ultrasound (FoCUS)
Point-of-care, standard but restricted protocol as an adjunct to physical examination by operator trained in FoCUS
Can use HUD devices
Operator trained in FoCUS
Sufficient to rule in/out major pathology
Targeted (goal-orientated) echocardiography
Answering a single, specific, often critical and frequently complex clinical question
Fully trained operator with access to all potential techniques
Streamlines diagnostic process and expedites interventions
CPR-compliant echocardiography
A subgroup of FoCUS used to rule in/out causes of cardiac arrest
Must be ALS-compliant
Sufficient to rule in/out major pathology
Critical care echocardiography
Application of echo within ICUs to help manage and guide clinical decision-making in critically ill
Frequently complex and encompasses all techniques
Requirement for specific training
Essential for monitoring complex cardiovascular patients, up to and including acute MCS

Figure 1 Definitions of cardiac ultrasound in cardiovascular emergency and critical care. ALS, advanced life support; CPR, cardiopulmonary resuscitation; FoCUS, focus cardiac ultrasound; HUD, handheld ultrasound device; ICU, intensive care unit; MCS, mechanical circulatory support.

further encompasses the role of CUS in cardiac critical care settings. This document includes a pathophysiological approach and experts' consensus on when and how to perform CUS assessments, the interpretation of findings, and their integration into the clinical decision-making process.

Pathophysiological approach to CUS in cardiovascular emergencies and critical care

Cardiogenic shock

The underlying pathologies leading to haemodynamic instability and/or cardiogenic shock (CS) vary widely (*Figure 2*). The widespread adoption of the Society of Coronary Angiography and Intervention classification together with the Shock Academic Research Consortium (SHARC) definition provides clarity regarding aetiologies and severity, specifically considering the causes of CS as acute myocardial infarction (AMI)-related, heart failure (HF)-related, secondary (due to another primary non-myocardial cardiac cause: arrhythmia, pericardial, or valvular disease), and post cardiotomy. Amongst those, additional key modifiers have been highlighted (modifiable or non-modifiable) as well as epidemiology, pathophysiology, aetiology, and presence/absence of cardiac arrest.

The roles of CUS in the patient pathway are:

- identify typical features of specific pathophysiology,
- quantify the severity of cardiac abnormalities at the time of investigation,
- improve the prognostic stratification, and
- monitor the cardiovascular changes according to management.

Acute myocardial infarction

Left ventricular (LV) ejection fraction (EF) is commonly used in assessing ventricular function in acute settings, but it has well-established limitations, particularly in asynchronous contraction and subtle myocardial ischaemia, where it lacks sensitivity. In the ischaemic cascade, functional changes precede electrocardiographic abnormalities, with diastolic and longitudinal function impairments occurring before circumferential wall motion abnormalities.[27] Basic echocardiographic parameters such as mitral annular plane systolic excursion (MAPSE) and tissue Doppler imaging (TDI; S′ velocity), along with transmitral Doppler flow, provide early insights into myocardial dysfunction. Additionally, post-ejection shortening may contribute to dyssynchrony and stroke volume reduction.[28] The Wall Motion Score Index (WMSI) is a valuable tool for assessing the extent of asynergy in AMI, aiding in prognosis, treatment decisions, and monitoring, though it is operator-dependant, influenced by loading conditions and other conditions (e.g. pre-existing cardiomyopathies).[29]

When regional wall motion abnormalities (RWMAs) are not clearly visible, myocardial strain imaging (deformation imaging) offers advanced

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Table 3 Summary of the appropriateness criteria for the use of cardiac ultrasound in cardiac and cardiac-like emergencies

Emergency clinical presentations	Causes[a]	Echocardiography appropriate[b,c]	Echocardiography not appropriate[c]
Acute chest pain	Frequent: ACS, AoD, PE, MP, Ptx Less frequent: ADHF, T, AVR/PVD	(1) Evaluation of acute chest pain in patients with suspected myocardial ischaemia or infarction and non-diagnostic ECG and cardiac enzymes, and when resting echocardiogram can be performed during the pain (2) Evaluation of acute chest pain in patients with known underlying cardiac disease (valvular, pericardial, or primary myocardial disease) (3) Evaluation of patients with chest pain and haemodynamic instability unresponsive to simple therapeutic measures (4) Evaluation of chest pain in patients with suspected AASs, PE, myopericarditis, and Takotsubo cardiomyopathy (5) As an initial imaging modality for diagnosis of suspected aortic dissection in the emergency setting (6) Guiding the therapeutic approach in patients with known PE (e.g. thrombectomy and thrombolytics) (7) In patients with suspected pericardial disease, including effusion, constriction, or effusive-constrictive process (8) In patients with suspected bleeding in the pericardial space (e.g. trauma, perforation, aortic, or myocardial rupture). (9) Guidance and monitoring of pericardiocentesis	(1) Evaluation of chest pain when non-cardiac cause is apparent (2) Evaluation of chest pain in patients with confirmed diagnosis of myocardial ischaemia or infarction (3) In patients with suspected PE to rule out the diagnosis (4) As an elective diagnostic strategy in haemodynamically stable, normotensive patients with suspected PE
Acute dyspnoea	Frequent: ADHF, PE, T, AVR/PVD, ACS Less frequent: AoD, PTx, MP	(1) Distinguishing cardiac vs. non-cardiac aetiology of dyspnoea in patients in whom clinical and laboratory findings are ambiguous (2) Assessment of LV size, shape, and global and regional function in patients with suspected clinical diagnosis of HF (3) Detection of echocardiographic signs of tamponade (4) Detection of acute valvular regurgitation and/or PV dysfunction (5) Detection of suspected complication of myocardial ischaemia/infarction, including but not limited to acute MR, ventricular septal defect, free-wall rupture/tamponade, RV involvement, HF	
Haemodynamic instability or shock	Frequent: ADHF, T, AVR/PVD, PE, ACS Less frequent: AoD, PTx, MP	(1) For differential diagnosis of the cause of hypotension or shock, by detecting cardiac or non-cardiac causes (2) Rapid identification of pericardial effusion, LV or RV dysfunction, and acute valvular dysfunction (3) Rapid assessment of intravascular volume status	(1) In patients in shock of apparently non-cardiac cause (e.g. anaphylactic, neurogenic, haemorrhagic)

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Table 3 Continued

Emergency clinical presentations	Causes[a]	Echocardiography appropriate[b,c]	Echocardiography not appropriate[c]
New murmur	Frequent: AVR/PVD, ACS, AoD Less frequent: MP, ADHF, PE	(1) In patients with cardiac murmurs and symptoms or signs of or suggestive of HF, myocardial ischaemia or infarction, syncope, thromboembolism, or infective endocarditis, or clinical evidence of structural heart disease (2) Detection of valvular vegetations indicating infective endocarditis	
Chest trauma	Frequent: T, AoD, PTx Less frequent: ACS, AVR/PVD	(1) Detection of pericardial effusion, myocardial contusion or laceration, RWMAs, acute valvular regurgitation, and aortic dissection in patients with severe deceleration injury or blunt or penetrating chest trauma	(1) Routine evaluation in the setting of mild chest trauma with no electrocardiographic changes or biomarker elevation
Cardiac arrest or CPR	Frequent: ACS, PE, T Less frequent: AoD, MP, AVR/PVD, PTx	(1) Identification of the reversible or unexpected cause of cardiac arrest to guide CPR and therapy (e.g. tamponade, PE, hypovolaemic heart, hypertrophic cardiomyopathy)	(1) As a routine/obligatory procedure during CPR, or if it interferes with CPR
Syncope	Frequent: AS, arrhythmias, ACS, PE Less frequent: T, HCM, severe PHT	(1) Evaluation of patients with unexplained syncope to assess for structural heart disease (e.g. severe AS, HCM) (2) Assessment of left and RV function in patients with syncope and suspected HF or myocardial ischaemia (3) Identification of pericardial effusion or tamponade in patients with syncope and hypotension (4) Detection of acute PE in cases of syncope with suspected RV dysfunction (5) Evaluation of AVR/PVD as potential causes of syncope	(1) Routine echocardiographic assessment in patients with syncope and a clearly established non-cardiac cause (e.g. vasovagal syncope with typical presentation) (2) Evaluation of syncope in patients with normal ECG, no history of heart disease, and no clinical suspicion of structural heart disease

ACS, acute coronary syndrome; ADHF, acute decompensated heart failure; AS, aortic stenosis; AoD, aortic dissection; AVR/PVD, acute valvular regurgitation/prosthetic valve dysfunction; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; HCM, hypertrophic cardiomyopathy; LV, left ventricular; MP, myopericarditis; PE, acute pulmonary embolism; PHT, pulmonary hypertension; PTx, pneumothorax; RV, right ventricular; T, cardiac tamponade.
Modified and reproduced with permission from Oxford University Press from Neskovic *et al.*[25]
[a]In case of chest trauma, consequences are listed, not causes.
[b]TOE is indicated when TTE is non-diagnostic.
[c]Appropriateness criteria related to specific conditions are applicable for all emergency clinical presentations patients may have.

assessment by detecting post-systolic shortening/thickening (*Figure 3*) that could potentially indicate ischaemia, distinguishing transmural from non-transmural infarction, and assessing myocardial viability and prognosis.[30]

Importantly, strain imaging can detect myocardial dysfunction even in cases where global LVEF remains preserved, making it particularly useful in conditions such as myocardial ischaemia and sepsis-induced cardiomyopathy. Additionally, strain imaging plays an important role in risk stratification after myocardial infarction.[31] The main determinants of global longitudinal strain (GLS) are intrinsic myocardial contractility, loading conditions (pressure/volume), geometry (dilatation/hypertrophy), fibrosis, and heart rate. GLS reflects global LV systolic impairment in AMI, and values >−15% early in the disease course are associated with worse short- and long-term outcomes.[32] However, GLS interpretation must be cautious, as strain values may be affected not only by coronary artery disease (CAD) but also by diabetes mellitus, hypertension, non-ischaemic cardiomyopathies, and significant valvular disease, and they tend to be lower in males and older individuals.[33] Furthermore, the application of strain imaging in critically ill patients is limited by time constraints, equipment availability, imaging quality, and operator skill.

Beyond strain imaging, total isovolumic time (t-IVT) has shown excellent sensitivity in detecting ventricular dysfunction, particularly in patients with reduced coronary reserve undergoing dobutamine stress echocardiography (SE)[34] and in those with CS (*Figure 4*). Notably, longitudinal function and t-IVT have emerged as strong predictors of successful weaning from veno-arterial extracorporeal membrane oxygenation (V-A ECMO) in patients with CS.[35]

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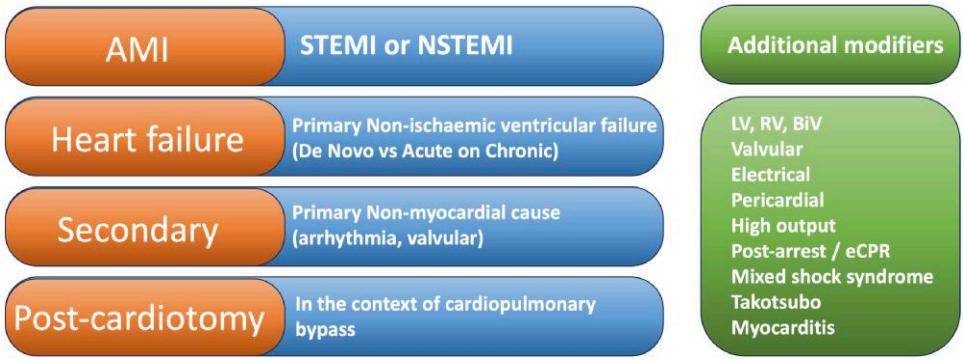

Figure 2 Summary of classifications of CS. Adapted from the existing SHARC framework for classifying types of CS. Modified from Waksman *et al.*[26] under the Creative Commons Attribution License 4.0 (CCBY): https://creativecommons.org/share-your-work/cclicenses/: AMI-CS, acute myocardial infarction-cardiogenic shock; BiV, biventricular; HF-CS, heart failure–cardiogenic shock; eCPR, extracorporeal cardiopulmonary resuscitation; STEMI, ST segment elevation myocardial infarction; LV, left ventricular; L, left; NSTEMI, non-RV, right ventricular; R, right.

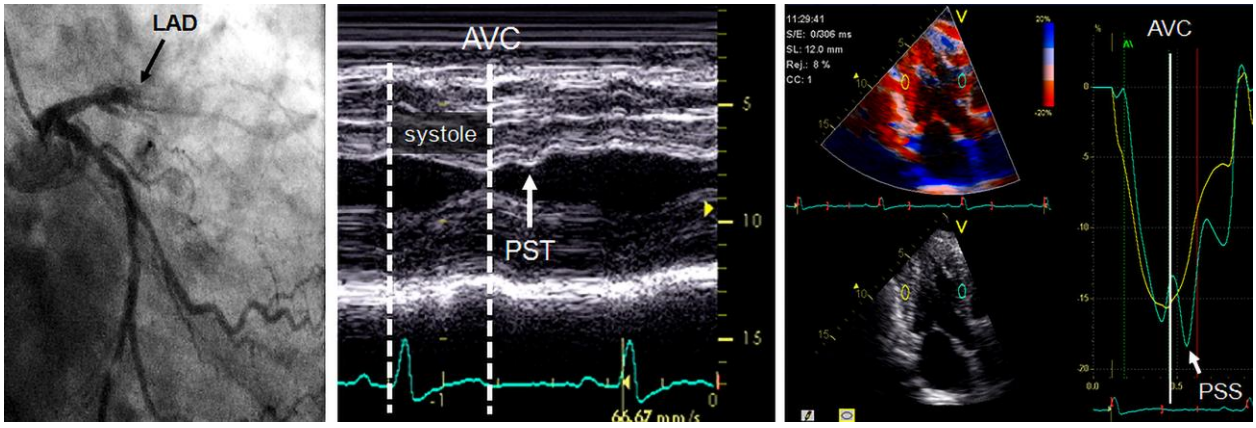

Figure 3 Altered deformation of the anteroseptal wall in a patient with suspected acute coronary syndrome without actual chest pain and with normal standard 2D echocardiogram. M-mode recording (middle panel) showing the 'double peak' contraction pattern of the septum (arrow pointing at post-systolic thickening of the septum, not detectable in the posterior wall). Tissue Doppler-derived segmental longitudinal strain curves (right panel) of normal posterior (yellow curve with a singlepeak occurring at aortic valve closure) and ischaemic anteroseptal wall (double peak green curve with the latter post-systolic peak occurring after the aortic valve closure). Coronary angiography (left panel) revealed a proximally occluded (arrow) left anterior descending coronary artery (LAD) with moderate collateral circulation. The goal is to assess suspected acute coronary syndrome without actual chest pain and normal standard 2D echocardiogram, and this requires advanced-level training in CUS. AVC, aortic valve closure; LAD, left anterior descending; PSS, post-systolic shortening; PST, post-systolic thickening.

The absence of hypercontractility in non-infarcted segments and the presence of remote dyssynergy may indicate multivessel CAD.[36–38] While LVEF assessment in the acute phase may not always reflect true functional loss, a reduced post-infarction LVEF remains a strong predictor of poor outcome.[29]

Right ventricular (RV) dysfunction in AMI is also linked to worse outcomes. Since assessing RVEF reliably can be challenging, alternative markers of longitudinal function, such as tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler-derived *S′* velocity, provide reproducible and reliable measures of RV contractility.[39] Additionally, RV free-wall longitudinal strain (RVfwLS) has been linked to HF and arrhythmias in patients with inferior-wall ST-segment elevation myocardial infarction (STEMI).[40] Moreover, RV GLS, measured 24 h after revascularization in first-time AMI patients, has been found to outperform conventional RV function indices in predicting in-hospital mortality.[41]

In the context of AMI, severe diastolic dysfunction and/or functional ischaemic mitral regurgitation (MR) may lead to a rise of left atrial pressure (LAP) and cardiogenic pulmonary oedema. Even mild functional MR detected within the first 48 h post-AMI is associated with worse prognosis.[42] Although $E/e' > 15$ is frequently used to estimate LV filling pressures, its correlation with pulmonary capillary wedge pressure (PCWP) remains suboptimal.[43] A more sensitive marker is left atrial strain (LA strain), which correlates with elevated LAP and adverse outcomes in AMI patients.[44]

However, as with many parameters in critical care echocardiography, trend analysis is often more valuable than absolute numbers.

Valuable prognostic markers in AMI include low LVEF,[45] extensive RWMA, i.e. high WMSI,[46] infarct expansion,[47,48] large LV end-systolic

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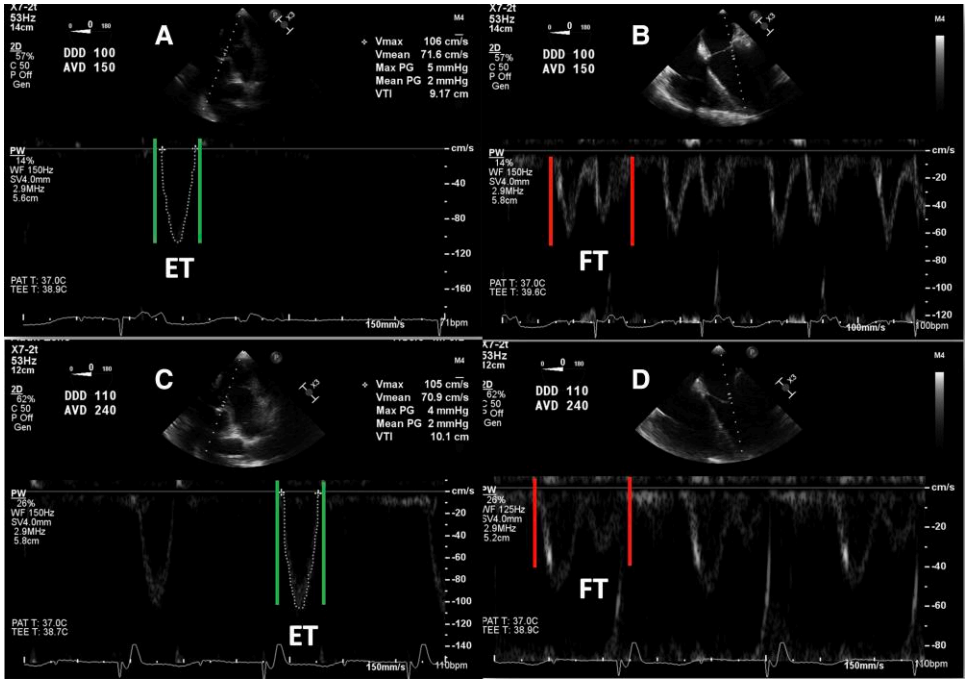

Figure 4 Echocardiography-guided cardiac output optimization using pulsed-wave Doppler estimation of t-IVT. (*A*, *B*) Tranasortic and transmitral pulsed-wave Doppler imaging at 100 bpm and atrioventricular delay of 150 ms. (*C*, *D*) Tranasortic and transmitral pulsed-wave Doppler imaging at 110 bpm and atrioventricular delay 240 ms. The filling time (FT) is measured from the start to the end of transmitral filling, and the ET from the start to the end of aortic ejection. The total ejection (t-ET) and filling (t-FT) periods are then derived as the product of the corresponding time interval and heart rate and expressed in s/min. t-IVT (also in s/min) is calculated as 60 – (t-FT + t-ET). A heart rate increase of 10 bpm resulted in a reduction of t-IVT from 16 to 13.9 s/min, and a corresponding increase in cardiac output from 2.6 to 3.2 L/min. AVD, atrioventricular delay; ET, ejection time; FT, filling time.

and end-diastolic volumes,[49] reduced GLS,[32] as well as diastolic parameters, such as short transmitral *E*-wave deceleration time,[50] restrictive transmitral flow pattern,[48] and septal $E/e' > 15$,[43] all of which are associated with worse short- and long-term outcome.

Another critical post-MI concern is the presence of LV thrombus, which is often associated with large anterior infarctions and severe LV dysfunction. LV thrombus increases the risk of systemic embolism and stroke, necessitating early detection and possible anticoagulation. While standard echocardiography can identify thrombi, myocardial contrast echocardiography (MCE) provides greater sensitivity, particularly in cases of apical foreshortening or poor acoustic windows, and offers additional insights into myocardial viability.[51] Observational data suggest that MCE use is associated with a reduced need for additional imaging [transthoracic echocardiography/transoesophageal echocardiography (TTE/TOE)] during hospitalization and shorter ICU stays.[52]

Despite the critical role of echocardiography in AMI, routine assessment at presentation must not delay revascularization[53] (*Figure 5*).

Key messages:

- CUS provides valuable information in all patients with CS due to AMI for diagnosis, functional assessment, detection of complications, and/or prognosis.
- Reduced longitudinal function, abnormal contraction pattern, RWMAs, or post-systolic shortening may represent early signs of perfusion abnormalities.

Mechanical complications of AMI

Echocardiography is the first diagnostic tool in the evaluation of mechanical complications of AMI.[54] Understanding the pathophysiology behind these complications allows for targeted echocardiographic examination.

Ventricular septal rupture

Ventricular septal rupture (VSR) creates a left-to-right shunt leading to RV volume overload and acute HF. Thus, RV function is crucial for maintaining haemodynamic stability.[55]

Acquired ventricular septal defects caused by ischaemic myocardial necrosis are often localized at the border of the asynergic zone.[56,57] They are of irregular shape and complex three-dimensional (3D) form, and, therefore, visualization may require 'multiple standards and modified', 'off-axis' echocardiographic views.[57,58] Colour Doppler or pulsed-wave Doppler mapping are essential, revealing 'systolic turbulent flow' across or on the RV side of the septum, indicating pathologic communication even in the case of visually 'preserved' continuity of the septum by 2D scan. Importantly, lower values of *trans*-ventricular gradient by continuous-wave Doppler (CWD) are associated with worse haemodynamic compromise,[58,59] while the presence of right-to-left shunt in diastole indicates severe RV dysfunction and predicts high mortality.[58]

Papillary muscle rupture

This condition results in acute MR due to the detachment of the papillary muscle from the infarcted area. Rupture of the posterior

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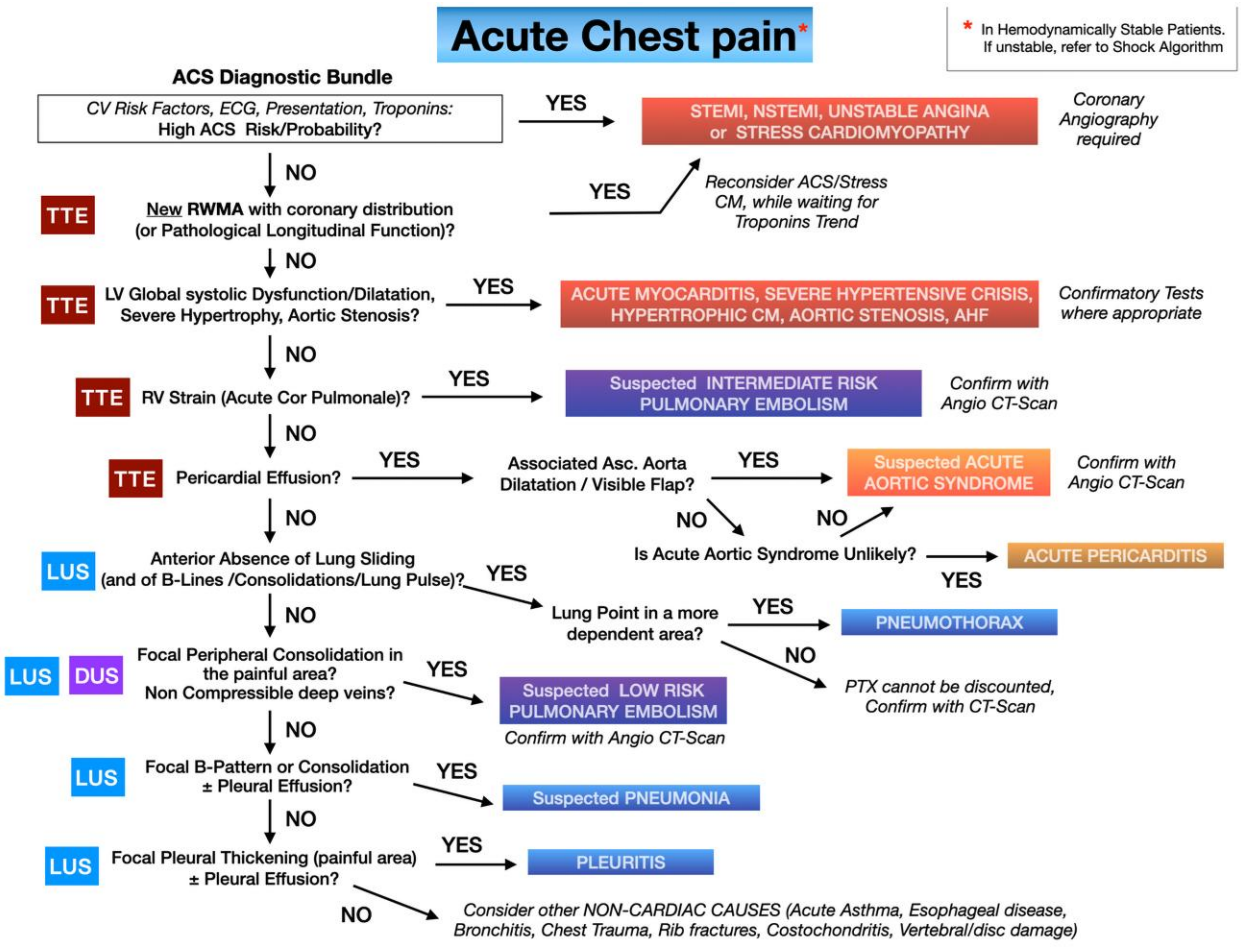

Figure 5 The figure shows a proposed algorithm for the CUS-guided evaluation of acute chest pain in acute settings (courtesy of WinFOCUS). Asc., ascending; ACS, acute Coronary Syndrome; AHF, acute heart failure; Cath Lab, catheterization laboratory; CV, cardiovascular; CM, cardiomyopathy; DUS, Doppler ultrasound; ECG, electrocardiogram; LUS, lung ultrasound; N, no; LV, left ventricle; RV, right ventricle; RWMA, regional wall motion abnormalities; TTE, transthoracic echocardiogram; Y, yes. Description of various LUS terminology mentioned in this figure can be found in the relevant EACVI LUS consensus document.[3]

papillary muscle is more common due to a single coronary artery supply and can be partial or complete. The pathophysiological consequence is severe acute MR, leading to pulmonary oedema and CS.[56,58,59]

'Complete rupture' shows flail leaflet(s), with an associated attached echogenic mass of the ruptured papillary muscle. In case of 'partial rupture', a piece of the papillary muscle is attached by chordal apparatus to the flail leaflets.

Colour Doppler may reveal various MR jet shapes and directions, and although regurgitation is always severe, it may underestimate the severity of regurgitation since due to tachycardia and rapid equalization of pressures between the two chambers. In addition, the large regurgitant orifice often results in more laminar than turbulent, lower velocity regurgitant flow, more difficult to delineate on colour Doppler and, therefore, the key echocardiographic finding for making diagnosis remains 2D imaging of the freely moving echogenic mass attached to flail leaflet(s). TOE may be essential to reach diagnosis in challenging cases and in critically ill patients on mechanical ventilation. The use of 3D echocardiography may offer further insights into the spatial relationships of the valve structures.[60]

LV free-wall rupture and pseudoaneurysm

'Acute free-wall rupture' results in haemopericardium, cardiac tamponade, electromechanical dissociation, and sudden cardiac death. In immediate survivors, CUS reveals pericardial fluid in the haemodynamically unstable patient with RWMA due to AMI, and echocardiographic signs of tamponade. The free-wall defect at the rupture site and flow across the defect towards the pericardial space can be rarely visualized while 3D echocardiography may provide incremental morphologic information.[61]

Importantly, patients may survive an acute free-wall rupture if it is contained by pericardial adhesions and isolated with thrombosis at the rupture site. Up to 40% of cases represent 'subacute' forms of free-wall ruptures, evolving over hours or even days[62]; during this period, patients may be without symptoms and clinically stable. When it occurs, a 'subacute rupture' is characterized by the presence of pericardial effusion and hypotension of various degrees of severity, with or without signs of tamponade.[63] Administration of a contrast agent can demonstrate active communication into the pericardium.[64] 'A high index of clinical suspicion' is needed, and clinical characteristics associated with increased risk of rupture should be carefully considered (e.g. first transmural MI in elderly patients, delayed hospital admission or late reperfusion, single vessel disease

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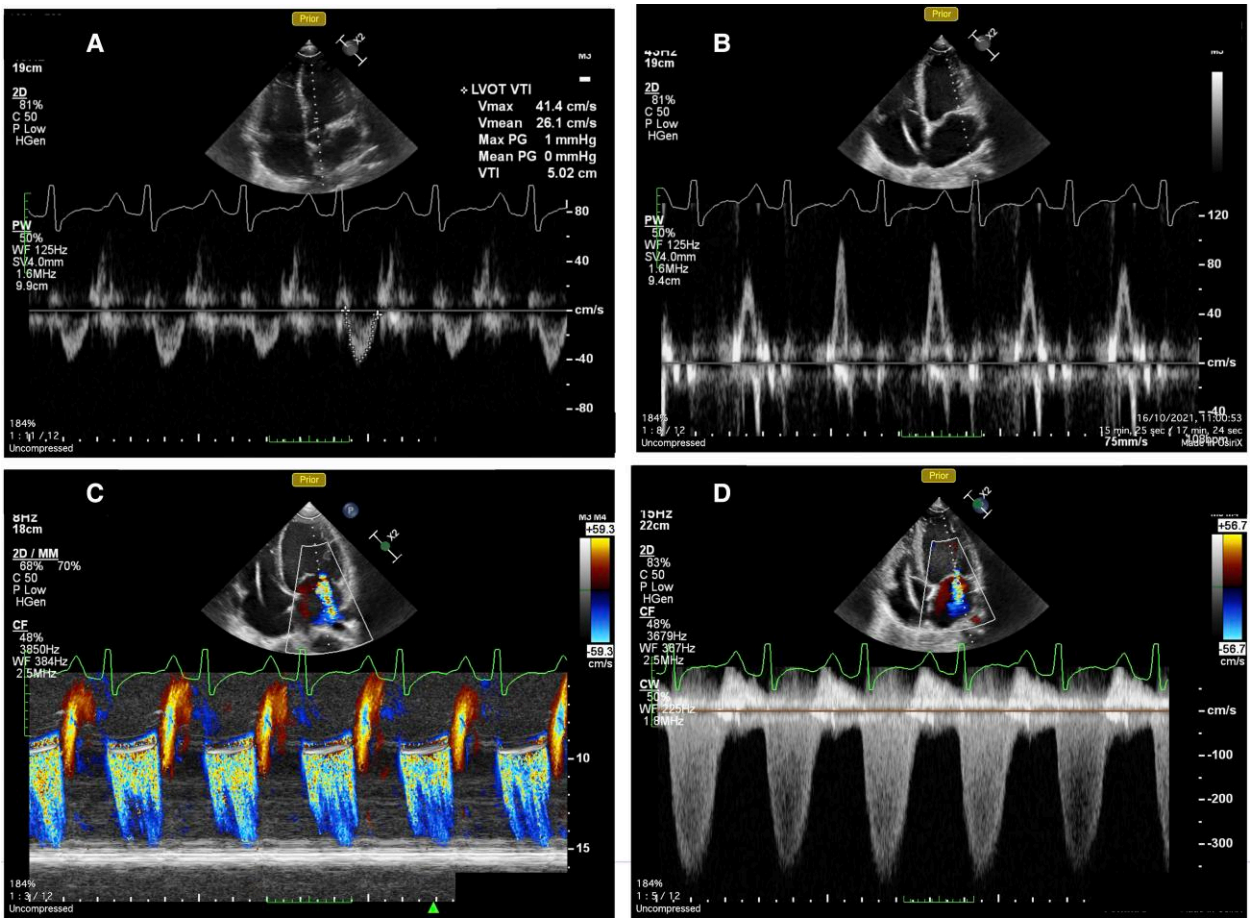

Figure 6 A patient with CS due to ADHF with the following Doppler haemodynamic features: (*A*) PWD of LVOT showing LVOT VTI of 5 cm, indicating stroke volume of 15.7 mL and estimated cardiac output of 1.7 L/min, (*B*) PWD of transmittal flow showing predominant filling during late active atrial contraction which indicates electromechanical dyssynchrony due to high LV diastolic pressure (LVEDP), (*C*) colour M-mode at the mitral valve level showing diastolic MR due to high LVEDP, (*D*) CWD at the mitral valve level showing a triangular dense CWD waveform indicating severe MR which extends into diastole. The goal is to assess haemodynamics of CS and this requires advanced level of training in CUS.

with poor collateral circulation, prolonged and recurrent chest pain, acute episodes of arterial hypertension, persistent ST-segment elevation, infarct expansion, and infarct extension).[63,65]

'Pseudoaneurysm' formation may occur in survivors of acute or subacute myocardial free-wall rupture if it is contained by pericardial adhesions and thrombus.[55,66,67] As opposed to true aneurysm, pseudoaneurysm typically reveals narrow communication (neck) with the LV cavity at the rupture site, with sharp discontinuity of the myocardium at the margins of the neck and absence of a myocardial layer in the pseudoaneurysmal sac. The wall of the pseudoaneurysm typically is composed of pericardium and clot.[55] Through the neck of pseudoaneurysm, bidirectional blood flow can be detected by colour Doppler during the cardiac cycle. Since a pseudoaneurysm requires 'urgent surgical repair' for prevention of spontaneous rupture, which occurs with high incidence,[55] it should be promptly differentiated from a true aneurysm.[67,68]

Key messages:

- CUS is the best bedside non-invasive test to detect mechanical complications of AMI, such as myocardial free-wall rupture, pseudoaneurysm, VSR, and papillary muscle rupture.
- It can accurately detect the cause of a new systolic murmur and/or haemodynamic instability in patients with AMI.

Acute decompensation of chronic HF

Congestion is a primary driver of outcome in CS related to HF; therefore, evaluation and severity assessment of cardiac and organ congestion are pivotal to direct management. Patients presenting with low cardiac output/CS due to *de novo* HF experienced a worse outcome when compared with those with acute decompensation of chronic HF (ADHF).[69,70] The differentiation of these two phenotypes relies mostly on demonstrating signs of chronic disease (e.g. dilatation of LV and atria, pronounced LV hypertrophy, severe valvular morphological abnormalities). In a large European multicentre survey, *de novo* acute HF patients showed a slightly higher mean LVEF compared with ADHF (42 vs. 36%).[71]

Comprehensive CUS evaluation may give detailed haemodynamic information (*Figure 6*). Comparing invasive (pulmonary artery catheterization) and 'echodynamic' evaluation of cardiac function in ADHF-related CS has confirmed excellent correlation in measuring cardiac index and output, cardiac power output (mean arterial pressure × CO/451), pulmonary artery pressures and low accuracy in PCWP evaluation (*Table 4*). Importantly, CUS is reliable in measuring pulmonary artery pulsatility index (peak TR pressure gradient)/estimated right atrial pressure), which has emerged as an important marker of RV function in the acute setting. In addition, RV contraction pressure

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Table 4 Correlation between non-invasive and invasive index indices in ADHF-CS[72]

Variable	Sensibility (%)	Specificity (%)	*R*	*P*-value
Cardiac index	97	73	0.88	<0.001
Systolic pulmonary pressure	87	63	0.91	<0.001
RAP[a]	90	83	0.86	<0.001
CPO	76	85	0.82	<0.001
PAPi	89	92	0.67	<0.001
PCWP[b]	84	55	0.34	0.01
API			0.40	0.017
RVCPI			0.66	<0.01
RAP/PCWP				>0.05

ADHF-CS, acute decompensated heart failure–cardiogenic shock; RAP, right atrial pressure; CPO, cardiac power output; PAPi, pulmonary pulsatility index; PCWP, pulmonary capillary wedge pressure; API, aortic pulsatility index; RVCPI, right ventricular cardiac power index.
[a]RAP is calculated as the integration of IVC diameter and collapsibility, hepatic venous flow pattern and tricuspid *E*/*e*′ ratio.
[b]PCWP calculated either as: 1.24 [*E*/*e*′] + 1.9[143]; and using the tricuspid regurgitation: TR peak gradient/2 in patients without known pulmonary arterial hypertension.

index (TAPSE × TR pressure gradient) as a proxy of RVSWI (stroke volume index × mean pulmonary artery pressure – mean right atrial pressure) × 0.0136, was able to differentiate different phenotypes (predominant left vs. right vs. biventricular).[72] The time difference between the onset of the *R*-wave on the electrocardiogram (ECG) and the *E*-wave of transmitral Doppler flow or *R*-wave at ECG and *E*′-wave at TDI is a reliable parameter of markedly increased LAP[73]; however, although this has a strong pathophysiological rationale, it has never been validated in the critically ill.

Considering the importance of multi-organ congestion in the HF-CS pathophysiology, the organ-specific ultrasound evaluation of congestion and perfusion is pivotal.[74] The venous excess ultrasound (VExUS) score has emerged as a promising multiparametric system incorporating inferior vena cava (IVC), renal and liver ultrasound with prognostic implications in HF patients.[5,75] However, different organ venous patterns may vary according to pathophysiology and predominant left or right failure.

The integration of lung ultrasound (LUS) and CUS significantly increases the diagnostic accuracy of assessment of congestion, and it also helps in prognostication.[3,4] Therefore, the two techniques should be concomitantly performed in critically ill (*Figures* 7 and 8). Indeed, the presence of 'multiple' (at least three B-lines in one chest zone), 'diffuse' (at least two positive zones per hemithorax), and 'bilateral' B-lines at LUS is highly specific for cardiogenic origin of respiratory failure.[76]

Key messages:

- Identifying signs of chronic cardiac disease in the context of HF-CS is pivotal to differentiate between ADHF and *de novo* acute HF.
- Doppler evaluation of cardiac output and pulmonary haemodynamics is reliable and allows identification of different phenotypes.
- Interrogation of venous filling pressure patterns may offer valuable information when experience is available and interpreted in the context of actual cardiac pathology.
- Integrating organ-specific ultrasound (CUS, LUS, and VExUS) enhances the assessment of congestion and perfusion in HF-CS, aiding both diagnosis and prognostication.

Acute severe MR

Acute severe MR may occur due to any of the following: mechanical complication following AMI (papillary muscle dysfunction, papillary muscle rupture), chordal rupture, endocarditis with valve destruction, mitral prosthetic dysfunction, systolic anterior motion (SAM) of the mitral valve, and functional MR on weaning from mechanical ventilation. TOE is superior to TTE in evaluating anatomic changes of the mitral apparatus and assessing the severity of MR, particularly in eccentric jets. In severe eccentric MR, the regurgitant jet may adhere to the LA wall, a phenomenon known as the Coandă effect, causing energy dissipation and appearing smaller and less turbulent on colour Doppler imaging, which can lead to underestimation of regurgitation severity.

Key Doppler indicators to observe in severe MR typically include[77,78]:

- The jet is usually eccentric in AMI-related mechanical complications.
- In many cases, such as prosthetic valve (PV) dehiscence, the jet width may not be easily visualized, but a large jet is typically evident.
- Normal LV global function or hypercontractility is frequently observed.
- A rare indicator of very severe MR is a more triangular spectral Doppler profile with rapid equalization of LA and LV pressures.

Key messages:

- The detection of a distinct structural abnormality of the mitral valve on CUS, such as a flail leaflet where the tip of the leaflet appears in the left atrium during systole, strongly suggests severe MR, even if the colour Doppler findings are unclear. Conversely, the absence of any visible morphological abnormality makes severe MR unlikely.
- In acute MR, the LV may remain normal in size with a hyperdynamic LVEF. Various 2D, colour, and spectral Doppler CUS techniques can be used to assess the size and severity of the MR jet and to illustrate the acute volume load on the left ventricle.
- TTE and TOE are essential for the perioperative management of patients with acute severe MR.

Acute severe aortic regurgitation

Acute severe aortic regurgitation (AR) is less common than acute MR, yet it remains a life-threatening cardiovascular condition with serious haemodynamic implications. Acute severe AR can result from acute aortic dissection, endocarditis, PV dysfunction, balloon aortic valvuloplasty, or trauma to the aortic valve. Patients with acute severe AR typically develop acute haemodynamic decompensation which could range from cardiogenic pulmonary oedema, CS to cardiac arrest. Typically, CUS reveals the LV is of normal size, and overall LV contractility is normal or hyperkinetic, unless there is coexisting LV disease. This contrasts with chronic AR which allows the heart chambers to adapt to the slowly increasing pressures, resulting in progressive LV dilation and systolic dysfunction. The haemodynamic changes in acute severe AR are more profound in patients with a non-compliant LV (e.g. left ventricular hypertrophy); therefore, these patients often sustain more rapid and profound haemodynamic deterioration.

CUS is essential in the identification of patients with acute AR and in understanding the aetiology of regurgitation. Colour Doppler examination of the aortic valve is the simplest method for determining the presence and severity of regurgitation.[78] The 'vena contracta', which is the narrowest part of the jet as it exits the regurgitant orifice, serves as an indirect yet accurate measure of the regurgitant aortic orifice and is relatively unaffected by flow.[78] For assessing AR via TTE, this is best visualized from the parasternal long-axis view. A 'vena contracta' width of >6 mm indicates severe AR.[79] A Doppler AR velocity profile with a

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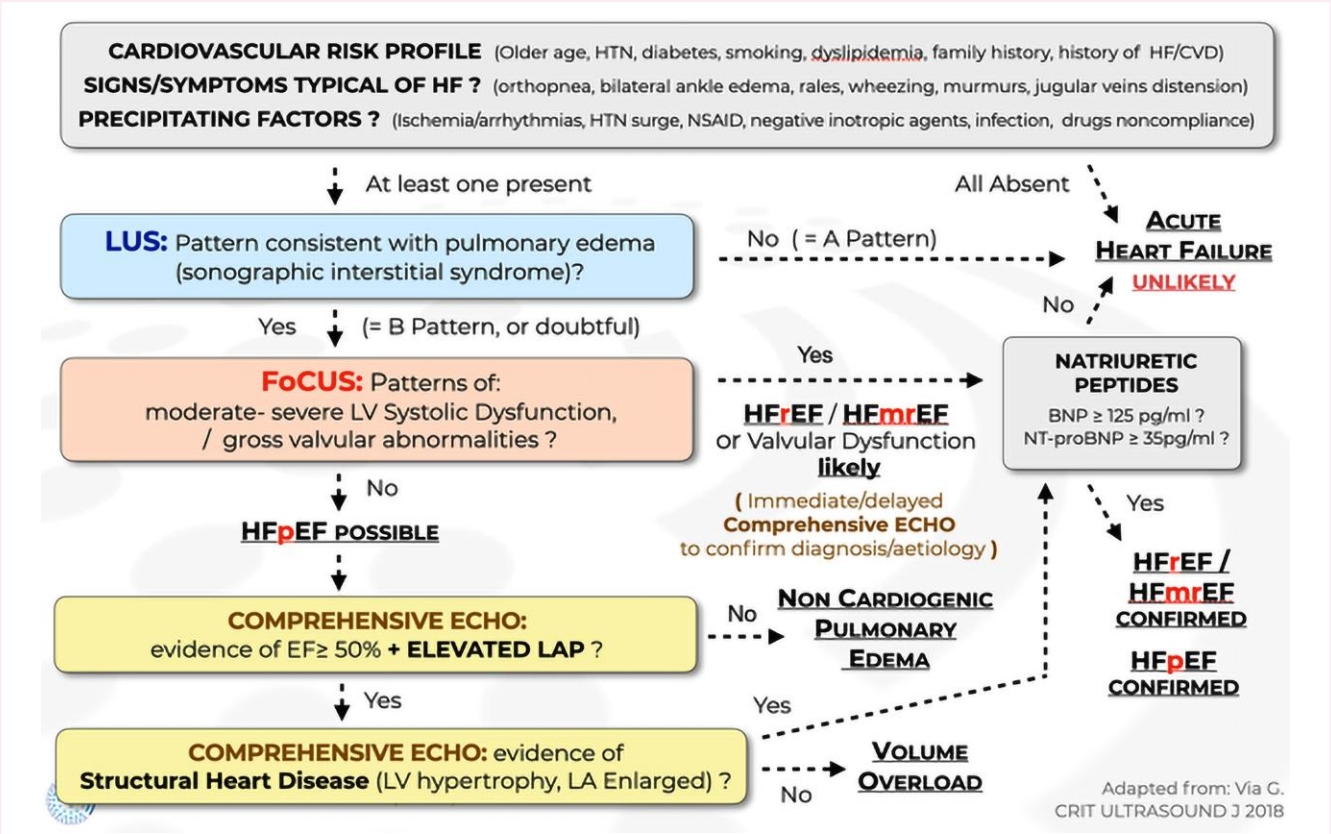
CARDIOVASCULAR RISK PROFILE (Older age, HTN, diabetes, smoking, dyslipidemia, family history, history of HF/CVD)
SIGNS/SYMPTOMS TYPICAL OF HF ? (orthopnea, bilateral ankle edema, rales, wheezing, murmurs, jugular veins distension)
PRECIPITATING FACTORS ? (Ischemia/arrhythmias, HTN surge, NSAID, negative inotropic agents, infection, drugs noncompliance)
At least one present
All Absent
LUS: Pattern consistent with pulmonary edema (sonographic interstitial syndrome)?
No (= A Pattern)
ACUTE HEART FAILURE UNLIKELY
No
Yes
(= B Pattern, or doubtful)
FoCUS: Patterns of: moderate- severe LV Systolic Dysfunction, / gross valvular abnormalities ?
Yes
HFrEF / HFmrEF or Valvular Dysfunction likely
(Immediate/delayed Comprehensive ECHO to confirm diagnosis/aetiology)
NATRIURETIC PEPTIDES
BNP ≥ 125 pg/ml ?
NT-proBNP ≥ 35pg/ml ?
No
HFpEF POSSIBLE
Yes
HFrEF / HFmrEF CONFIRMED
HFpEF CONFIRMED
COMPREHENSIVE ECHO: evidence of EF≥ 50% + ELEVATED LAP ?
No
NON CARDIOGENIC PULMONARY EDEMA
Yes
Yes
COMPREHENSIVE ECHO: evidence of Structural Heart Disease (LV hypertrophy, LA Enlarged) ?
VOLUME OVERLOAD
No
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Figure 7 CUS and LUS combined approach in the assessment of dyspnoea (courtesy of WinFOCUS). BNP, brain natriuretic peptide; CVD, cardiovascular disease; FoCUS, focus cardiac ultrasound; HTN, hypertension; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction; HRfEF, heart failure with reduced ejection fraction; LUS, lung ultrasound; LAP, left atrial pressure; LV, left ventricular, LA, left atrium NSAID, non-steroidal anti-inflammatory drugs; NT-proBNP, N-terminal brain natriuretic peptide.

dense spectral signal and a short pressure half-time (<200 ms) typically indicates haemodynamically severe acute AR. A short pressure half-time, however, while quite specific, is not sensitive for severe AR.[79] In cases where TTE windows are technically challenging, or with a severely eccentric aortic regurgitant jet, colour Doppler may underestimate the severity of acute AR. Due to the increased forward flow through the aortic valve, continuous-wave Doppler may show elevated transvalvular velocities. If the aortic valve appears without calcified, thickened leaflets or restricted opening of the native or prosthetic leaflets, these elevated transvalvular velocities are likely caused by AR rather than indicative of valvular stenosis. In this situation, a dense continuous-wave Doppler regurgitant jet with a short pressure half-time, combined with an elevated LV outflow tract (LVOT) velocity seen on pulsed-wave Doppler, may suggest acute severe AR, especially in a tachycardic patient with corresponding clinical symptoms. Holodiastolic flow reversal in the descending thoracic aorta (viewed from the suprasternal notch window on TTE or descending aorta view at 90° on TOE; *Figure 8*) and in the abdominal aorta (viewed from the subcostal window) is also indicative of severe AR.[78]

Key messages:

- In acute AR, the LV may remain normal in size with a hyperdynamic LVEF. Various 2D, colour, and spectral Doppler echocardiographic techniques can be used to evaluate the severity of the AR jet and to assess the acute volume load on the left ventricle.
- TTE and TOE are essential for the perioperative management of patients with acute severe AR.

PV thrombosis

PV obstruction remains a serious cause of high morbidity and mortality despite advances in diagnosis and management.[80] PV thrombosis is twice as likely to happen in the mitral than the aortic position independent of the type of PV. Echocardiography should focus on the assessment of the movement of valve occluders and estimation of Doppler gradients across the valve. Echocardiography involves the assessment of the mechanism of valve obstruction (thrombus vs. pannus) as well as assessment of cardiac function and haemodynamic consequences (ventricular function, cardiac output and filling pressures). TTE is the first-line imaging method in suspected acute PV thrombosis, yet in the context of CS and in mechanically ventilated patients' TOE might be superior. PV obstruction is suspected when transvalvular gradients measured with CWD are higher than the normal gradient of that PV model. If possible, it is important to compare gradients estimated in acute conditions with previously estimated gradients in the same patients in stable condition. While a new onset of an elevated mean gradient is typically indicative of PV thrombosis, up to 20% of patients with mitral PV thrombosis (excluding those with non-obstructive cases) may exhibit a normal Doppler gradient at rest.[81,82]

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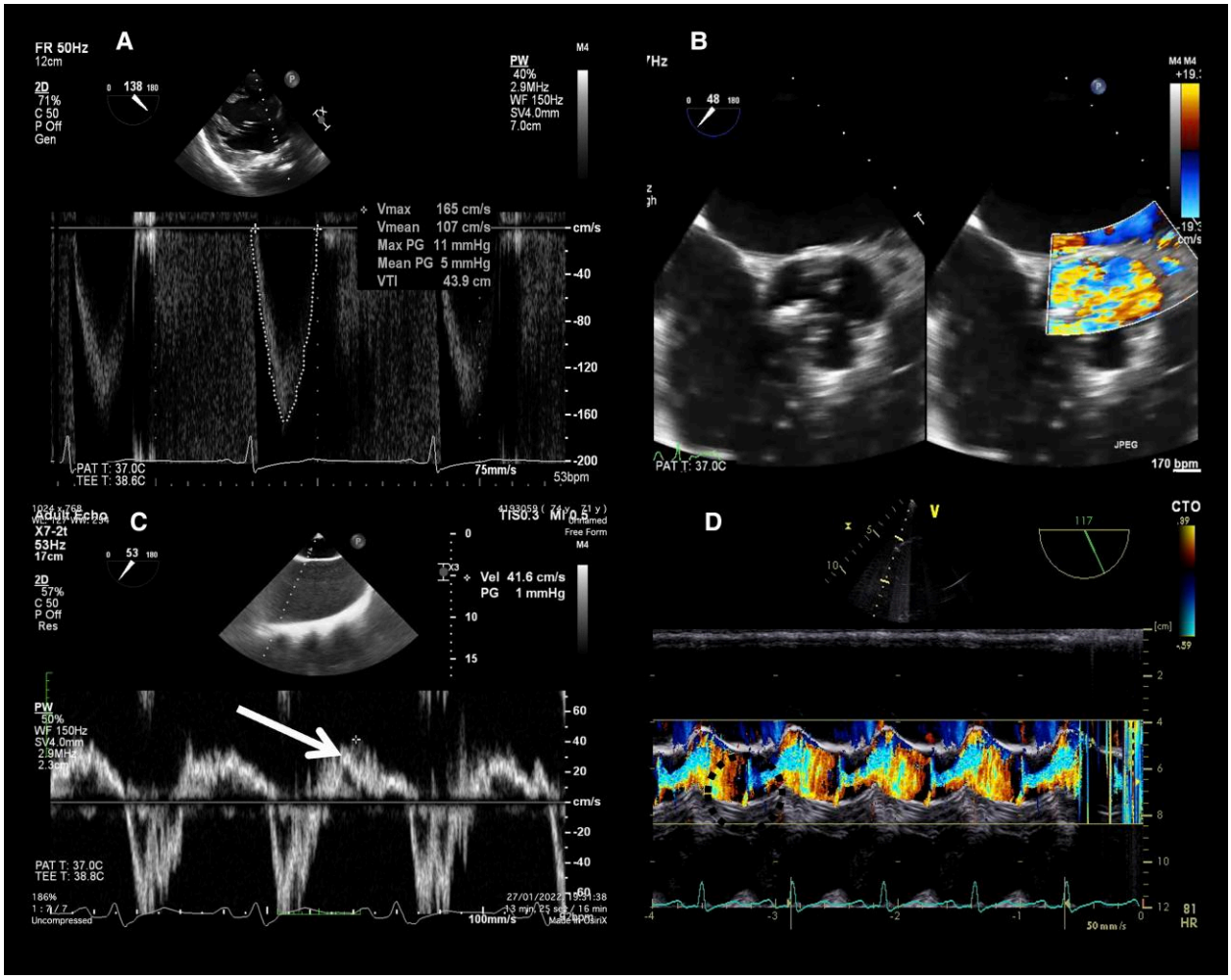

Figure 8 TOE in acute severe AR due to aortic valve endocarditis. (*A*) Mid-oesophageal long-axis view at 138° is showing an LVOT VTI of 43.9 cm which corresponds to an estimated stroke volume of 137.8 mL. This is overestimated due to severe AR which leads to increased LV end-diastolic volume; therefore, LVOT VTI is unreliable in estimating actual stroke volume in this case. (*B*) Mid-oesophageal RV inflow-outflow view at 60° is showing a broad jet of severe AR by colour Doppler. (*C*) Mid-oesophageal descending aorta long-axis view is showing diastolic flow reversal (white arrow) in the descending aorta which also denotes severe AR. (*D*) Colour M-mode at the aortic valve in mid-oesophageal long-axis view is showing colour M-mode Doppler imaging showing AR with interruption of regurgitation at mid-systole and presence of regurgitation into late systole. All of these findings were the result of equalization of LV and aortic pressures because of markedly elevated LVEDP caused by AR. The goal is to assess haemodynamics of acute severe AR and this requires advanced level of training in CUS.

Suggested stepwise approach is the following: (i) Assess valve mobility, evaluating the motion of the PV is the first step, particularly in bileaflet prostheses in the mitral position, where restricted leaflet motion can be a concern. TTE and/or TOE are fundamental for assessing disc motion and identifying potential obstructions. (ii) Check for gradients (flow-dependent), however, in small aortic PVs, gradients may appear overestimated, which can mimic obstruction despite a normally functioning valve. (iii) Calculate Effective Orifice Area (EOA) or Doppler Velocity Index (DVI) (flow-independent) to distinguish true obstruction from patient-prosthesis mismatch, calculating EOA or DVI provides a flow-independent assessment of PV function. (iv) Integrate with clinical context including anticoagulation status (e.g. INR in mechanical valves), symptoms, and the timing of the event, to differentiate true obstruction from pseudo-obstruction in PVs. Of note, direct visualization of leaflet motion in mechanical aortic prostheses is challenging by both TTE and TOE. Further echocardiographic parameters have been suggested to distinguish aortic valve prosthetic obstruction (AVPO) from a high functional gradient or patient-prosthesis mismatch. Key parameters are the delayed peak systolic velocity, which results in an extended acceleration time (AT) and a higher AT-to-ejection time ratio (AT/ET).[80] An AT >95 ms and an AT/ET ratio exceeding 0.32 have shown the highest accuracy (87–94%) in identifying AVPO.[83,84] AT, ET, AT/ET ratio, and dimensionless velocity index (the ratio of the subvalvular velocity obtained by pulsed-wave Doppler (PWD) and the maximum velocity obtained by CWD across the PV) are flow-independent parameters, and they should be combined with flow-dependent parameters in evaluating PV function independent of variability in cardiac output. CUS particularly TOE, plays a crucial role in guiding acute thrombolysis regimens by assessing thrombus size, mobility, and obstruction severity. It helps in determining thrombus resolution and detecting complications such as valve dysfunction during treatment.

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Key messages:

- The hallmark of PV obstruction by echocardiography is the limited or absent leaflet motion accompanied by an elevated mean pressure gradient measured by CWD.
- TOE remains superior to TTE in assessing occluder motion of mechanical PVs, especially in mitral position.
- Detection of the mechanism of valve obstruction (thrombus vs. pannus) in majority of cases is not possible by echocardiography.

PV dehiscence/regurgitation

All mechanical PVs and nearly all bioprosthetic valves exhibit minor 'signature jet' regurgitation even when functioning normally. In mechanical prostheses, a small amount of regurgitation occurs due to closure backflow during the occluder's (disc or leaflets) closing motion, and additional leakage flow can occur after closure due to small, intentional gaps between the occluder and the struts or the rings. These gaps are designed to ensure mobility and continuous flushing to prevent microthrombi formation. These minor physiological leaks are detectable on spectral and colour Doppler and produce regurgitant jet patterns specific to each valve type, but they can typically be easily distinguished from significant pathologic regurgitation. In bioprostheses, minimal regurgitation is also present, similar to the 'physiologic' regurgitation seen in native valves. Severe and often acute regurgitation in a valve prosthesis usually results from the following mechanisms: structural damage with embolization of the occluder after strut fracture, ring dehiscence and large paravalvular leaks, occluder being fixed in semi-open or semi-closed position by thrombosis or pannus, infective endocarditis. The clinical consequences of acute severe prosthetic regurgitation are similar to those of acute native valvular regurgitation and may result in CS. Recent advancements in 3D TOE have significantly enhanced the imaging of both mechanical and biological prosthetic leaflets. The techniques used to assess prosthetic regurgitation are fundamentally similar to those for native valves. However, the presence of 'artefacts and shadowing' from valve prostheses, combined with frequently altered cardiac morphology in these patients, makes the assessment more challenging. It's important to note that shadowing from a mitral prosthesis often obscures prosthetic regurgitation when viewed from an apical TTE window. The presence of a large proximal convergence zone on the ventricular side of the prosthesis, which is unobstructed, may suggest paravalvular leakage. Therefore, the use of parasternal and subcostal windows is essential, and TOE is advised. Although TOE is superior for assessing MR, evaluating AR (e.g. after transcatheter aortic valve replacement—TAVR) with TOE can be challenging, partly due to the difficulty in aligning the continuous-wave Doppler cursor with the direction of regurgitant flow. However, TOE can still provide valuable additional information, such as identifying vegetation from infective endocarditis or an abscess. Whenever PV thrombosis/dysfunction is suspected, expert echocardiographic assessment is needed.

Key messages:

- When PV malfunction or endocarditis is suspected, it is crucial to promptly perform a TTE and/or TOE examination.
- TOE is superior to TTE in the diagnosis and quantification of acute prosthetic valvular dehiscence/regurgitation, especially in mitral valve prosthesis.

Mixed cardiogenic and vasoplegic shock

Mixed CS (CS with at least 1 additional contributing cause of shock state) is the second most common form of shock in contemporary cardiac ICUs.[85,86] It is characterized by relative hypovolaemia related to low systemic vascular resistance (SVR) associated with ongoing sepsis/septic shock[87] or a SIRS response related to cardiopulmonary bypass or to CS itself.

In mixed CS related to sepsis, echocardiographic features vary according to the disease stage. At the initial phase where clinical phenotype is predominantly characterized by vasoplegia, the echocardiographic features, in the absence of pre-existing cardiac disease, are represented by 'signs of severe hypovolaemia' (small cardiac cavities and small IVC with respiro-phasic variations) and LV and RV (biventricular) hyperkinesia'. Cardiac dysfunction may occur within 72 h since the septic onset. Assessment of longitudinal function (either MAPSE or GLS) appears more accurate in detecting systolic dysfunction than LVEF[88,89] and prognostic stratification.[88] Diastolic dysfunction occurs very often, and it is significantly associated with mortality.[90] A simplified approach based on septal $e' < 8$ and $E/e' <$ or ≥ 13 has shown to be accurate in diagnosing diastolic dysfunction in sepsis and septic shock.[91] RV dysfunction (detected by TAPSE and/or RV GLS) is associated with worse prognosis.[92] Cardiac dysfunction during sepsis/septic shock is usually reversible in 7–10 days. Therefore, a daily assessment may be useful to guide management and stratify prognosis. A non-invasive method for evaluating SVR has been proposed [SVRi (dynes.s.m^2/cm^5) = (Mean arterial pressure − Right atrial pressure) (mmHg) × 80/cardiac index (L/min m^2), demonstrating a promising correlation with invasive measurements.[93]

Studies aiming to validate indices to estimate volume conditions (IVC, SVC) have selectively excluded patients with acute CVDs; therefore, these parameters should not be routinely applied in case of pre-existing structural cardiac disease or in CS patients. Dynamic variation of LVOT velocity time integral (VTI) during passive leg raising (LVOT VTI increase > 15–20%) may suggest preload responsiveness,[94] although no data exist specifically in a CS population. Here, when a super-imposed vasoplegic component is suspected, comprehensive haemodynamic evaluation with pulmonary artery catheter may be helpful. LVOT VTI variability using passive leg raising test is the only validated test in spontaneously breathing patients and in those with irregular heart rhythm.[95] Other tests of preload responsiveness require well-defined conditions (passive mechanical ventilation, 8 mL/kg tidal volume and sinus rhythm).[96]

Key messages:

- In mixed shock, an incremental worsening of cardiac function may occur on top of primary CS.
- Comprehensive assessment is essential, including CUS evaluation of global cardiac function, longitudinal myocardial function, and non-invasive SVR.

Obstructive shock

Obstructive shock may occur due to increased extra-myocardial pressure (cardiac tamponade), tension pneumothorax, acutely increased RV afterload, and LVOT obstruction.

Cardiac tamponade

Cardiac tamponade is a clinical diagnosis, while CUS may detect tamponade physiology and the haemodynamic significance of pericardia effusion, confirming diagnosis, evaluating aetiologies and guiding management.

During inspiration, an exaggerated increase in RV filling occurs at the expense of diminished LV filling (interventricular septum shifts to the left), which can be appreciated on pulsed Doppler as an increase in tricuspid and decrease in mitral inflow. Expiration is associated with reciprocal changes.

Cardiac tamponade occurs when there is a significant compression of the heart chambers by accumulation of abnormal pericardial contents (fluids, blood, clots, pus, and gas, separately or in combination). As the chambers become progressively smaller and myocardial diastolic compliance is reduced, cardiac inflow becomes limited, ultimately equalizing mean diastolic chamber pressures. Cardiac tamponade

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should always be seen as a *continuum* of events: even small elevations of intrapericardial pressure in the early phase of cardiac compression may have some impact on ventricular filling, and echocardiographic signs may be present even in the absence of large pericardial effusion and/or overt clinical tamponade. In patients with intravascular fluid depletion, cardiac compression and tamponade may occur with comparatively low pericardial pressure (low-pressure tamponade).

The echocardiographic warning signs of cardiac tamponade include pericardial effusion, late diastolic or early systolic right atrial collapse (earliest sign), diastolic RV collapse (high specificity), exaggerated inspiratory variation of the two ventricles (inspiratory expansion of the RV and simultaneous compression of the LV by the shift of the intraventricular septum to the left; reciprocal changes occur in the expiratory phase) that can be detected by PWD (decrease in transmitral inflow *E*-wave velocity of >25% on inspiration and/or increase in tricuspid inflow *E*-wave velocity by >40% on inspiration), and a plethoric IVC with minimal respiratory variations (high sensitivity). Importantly, the presence of both RA and RV collapse always indicates that the effusion is haemodynamically significant Importantly, RV hypertrophy and high RV intracavitary pressures (e.g. in patients with long-standing pulmonary hypertension) may prevent the occurrence of these collapses.

If a large amount of pericardial effusion is seen, the heart might be seen moving within the pericardial space ('swinging-heart'), but importantly, tamponade may occur even in cases when small amount of pericardial fluid accumulates rapidly (i.e. during percutaneous interventional procedures). However, early after cardiac surgery, cardiac tamponade may not be associated with classical echocardiographic features and can present with a localized (clot) rather than global pericardial effusion,[97] often in contrast with actual profound haemodynamic disturbance. These 'iatrogenic' effusions may be difficult to explore, and their detection often requires off-axis TTE views or TOE.

Finally, CUS can be successfully used for echo-guided pericardiocentesis.[98]

Key messages:

- Cardiac tamponade is always a clinical diagnosis supported by CUS, revealing warning signs of tamponade physiology.
- Presence of small pericardial effusion does not exclude diagnosis of tamponade.
- Diagnosis of cardiac tamponade after cardiac surgery can be challenging even with TOE, requires high index of suspicion and remains a clinical diagnosis.

Tension pneumothorax

The in-depth discussion of LUS is outside the scope of this document; however, pneumothorax needs to be ruled out by LUS[99] or other imaging modalities as a part of the overall assessment and diagnostic approach of shock states.

Increased RV afterload

Acute elevation of RV afterload may occur due to either haemodynamic [pulmonary embolism (PE) or respiratory triggers (pneumonia, adult respiratory distress syndrome (ARDS)]. In acute PE, the sudden increase in afterload leads to compensatory RV dilatation but also increases myocardial oxygen demand in the context of impaired coronary perfusion and hypoxaemia,[100] thus contributing to acute RV failure. CUS is recommended by the ESC guidelines on acute pulmonary embolism in the diagnosis and to guide the treatment of high-risk PE with haemodynamic instability if the patient is too unstable to undertake a computed tomography (CT) pulmonary angiographic study or if the facilities are not available within a consistent time frame.[101]

Established landmarks for RV evaluation and severity stratification include RV size, function and pulmonary haemodynamic evaluation. RV dilation (RV/LV > 1) with leftward interventricular septum bulging indicates a severe increase in RV afterload. RV dilatation measured at the apical four-chamber view with normal right atrial size is a sensitive marker of acute RV dysfunction. The measures of both TAPSE, RV *S′* with TDI and RV free-wall longitudinal strain reflect longitudinal function (cut-off in PE respectively, <15 mm, 10 cm/s, and −20%) having good correlation with global RV performance. In the presence of RV free-wall dyskinesia/akinesia preserved contractility of the RV apex has a moderated sensitivity (McConnell's sign). A ratio of TAPSE/PASP < 0.4 (RV/PA uncoupling) may predict deterioration in patient with high-risk PE.[102] Similarly, the ratio between sPAP and LVOT VTI, by providing simultaneous information on both right and left heart performance in response to increasing afterload, seems to be strongly associated with adverse events in high-risk PE.[103] The measurement of pulmonary valve AT < 60 ms along with an sPAP < 60 mmHg from a tricuspid regurgitation jet ('60/60 sign') using CWD has a sensitivity of 36% and specificity of 94% in diagnosing acute PE. In this situation with severe TR, the TR peak systolic velocity should not be used to estimate sPAP since it will underestimate it significantly and the calculated sPAP may be normal or mildly elevated in spite of high pulmonary vascular resistance (*Figure 9*).

Key messages:

- Echocardiography is valuable for guiding the treatment of high-risk PE if the patient is too unstable to undertake a CT angiographic study.
- RV size and systolic performance should be performed to stratify the severity of haemodynamic alterations.
- Doppler-derived systolic right heart pressures are reliable unless severe RV failure with severe TR, when sPAP may be severely underestimated.

LVOT obstruction

LVOT obstruction may occur in the case of severe hypovolaemia along with SAM or mid-cavity obstruction or may be related to structural abnormalities (hypertrophic cardiomyopathy, subaortic stenosis) or Takotsubo syndrome (TTS; apical ballooning morphology). CUS can be used to define the extent of septal bulging, mid-cavity flow acceleration, and CWD allows the quantification of LVOT systolic gradient by identifying an increased late-peaking systolic ('dagger-shaped' CWD waveform.[104] In dynamic obstruction, MR is variable and frequently due to associated SAM of the mitral valve. The MR jet due to SAM is eccentric, directed to the posterolateral wall of the left atrium (*Figure 10*).

Key message:

- In the clinical context of haemodynamic instability colour Doppler evidence of the presence of LVOT turbulence along with high-transvalvular aortic gradient or structural and functional abnormalities predisposing to LVOT obstruction (in case of severe LV systolic dysfunction) should prompt a change in management to optimize the preload and reduce LV hypercontractility/tachycardia.

Special clinical conditions

Peripartum cardiomyopathy

Peripartum cardiomyopathy (PPCM) is a rare but serious condition characterized by HF in the last month of pregnancy or within 5 months postpartum, in the absence of other identifiable causes of cardiac dysfunction.[105,106]

There are no pathognomonic CUS features of PPCM; however, it is usually associated with a global reduction of LV systolic function, dilated LV, and concomitant alteration in diastolic filling. Functional MR could also be observed, and RV dysfunction is a significant prognostic factor.[107] Abnormal GLS can be detected even when LVEF is only mildly reduced, offering an early indication of myocardial dysfunction.[108] Serial CUS within the clinical context helps differentiate PPCM from other causes of HF that may present during the peripartum period,

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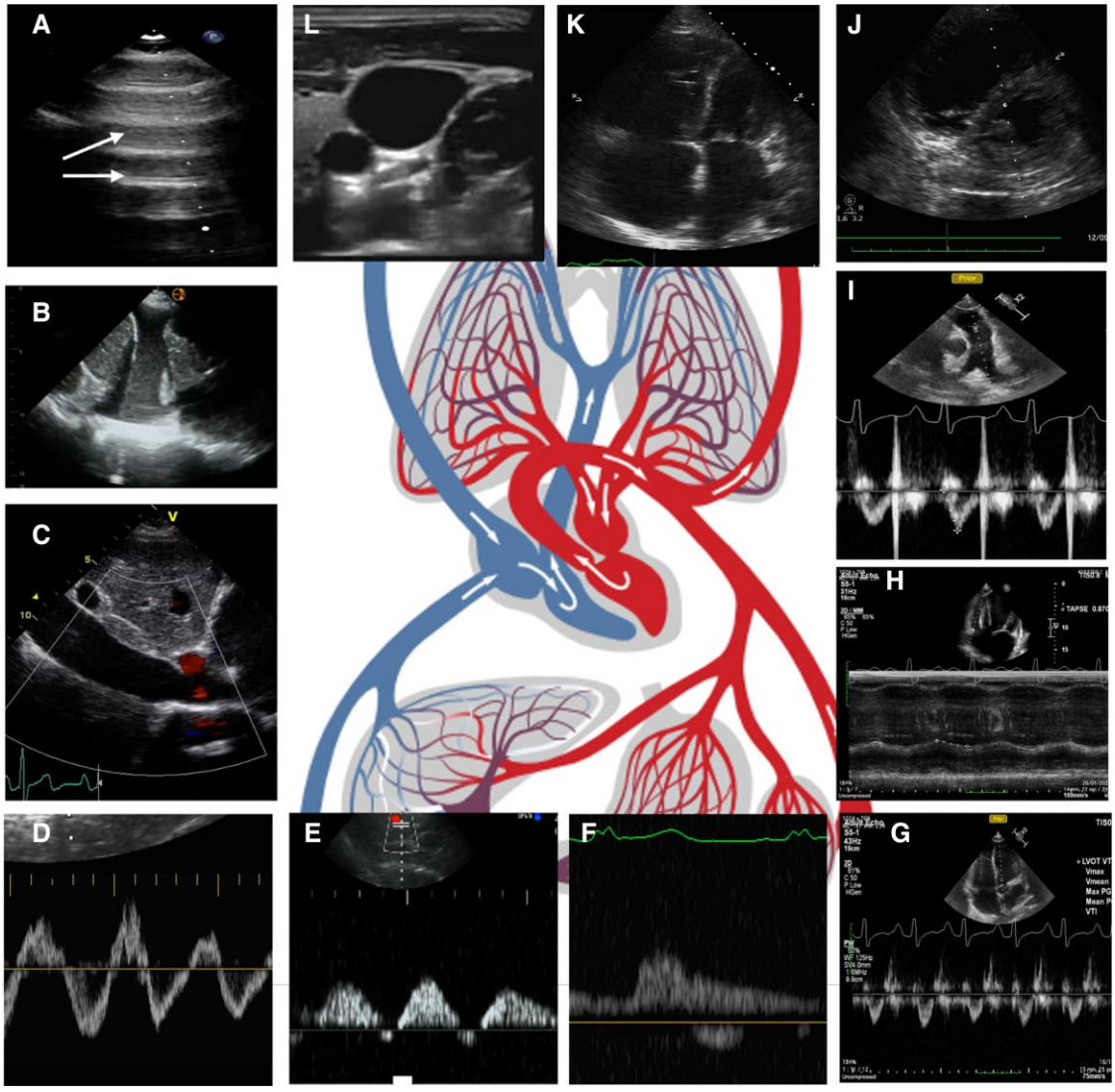

Figure 9 An illustration of multi-organ point-of-care ultrasound in a patient with obstructive shock due to acute PE which includes the following: (*A*) lung parenchymal ultrasound at the anterior zones showing absence of congestion (A-lines), (*B*) lung parenchymal ultrasound at the right lung base showing lobar consolidation with pleural effusion, (*C*) *trans*-hepatic mid-axillary view showing dilated IVA, (*D*) PWD of hepatic venous flow showing systolic reversal, (*E*) PWD of portal vein showing pulsatile flow, (*F*) PWD of renal arterial and venous flow showing isolated flow during diastole with no flow seen during ventricular systole, (*G*) PWD of LVOT flow showing severely reduced LVOT VTI and reduced stroke volume, (*H*) M-mode showing TAPSE < 10 mm at RV-focused apical four-chamber view, (*I*) left parasternal short-axis view at the level of great vessels showing PWD of RVOT, (*J*) left parasternal short-axis view showing D-shaped LV with a flat IVS, (*K*) apical four-chamber view showing dilated RV which is larger than LV, (*L*) 2D image of a dilated internal jugular vein. The goal is to assess haemodynamics of obstructive shock due to acute PE and this requires advanced level of training in CUS.

such as hypertensive heart disease, spontaneous coronary artery dissection, or pre-existing cardiomyopathies.[109]

Key message:

- Global hypokinesia and LV dilatation with/without acute valvular dysfunction are, although non-specific, suggestive of PPCM in the appropriate clinical context.

Acute myocarditis

Acute myocarditis can occur due to infectious and non-infectious causes with viral myocarditis being the most common type.[110] The clinical presentation varies in severity from mild symptoms to CS. Occasionally, the pericardium can be involved leading to myopericarditis with or without pericardial effusion.[111] International guidelines recommend that all patients with clinically suspected myocarditis should undergo a TTE at initial presentation.[112] However, echocardiography has low sensitivity so it cannot rule out myocarditis. CUS may reveal LV dilatation, reduced LV function, increased wall thickness due to myocardial oedema, RWMAs, diastolic dysfunction, an elevated sphericity index, and signs of concurrent pericardial involvement.[113,114] Fulminant myocarditis is often characterized by a smaller ventricular cavity and a mild increase in wall thickness resulting from myocardial oedema.[112] LV GLS has incremental diagnostic and prognostic value but lacks specificity, as similar alterations occur in other HF conditions.[115]

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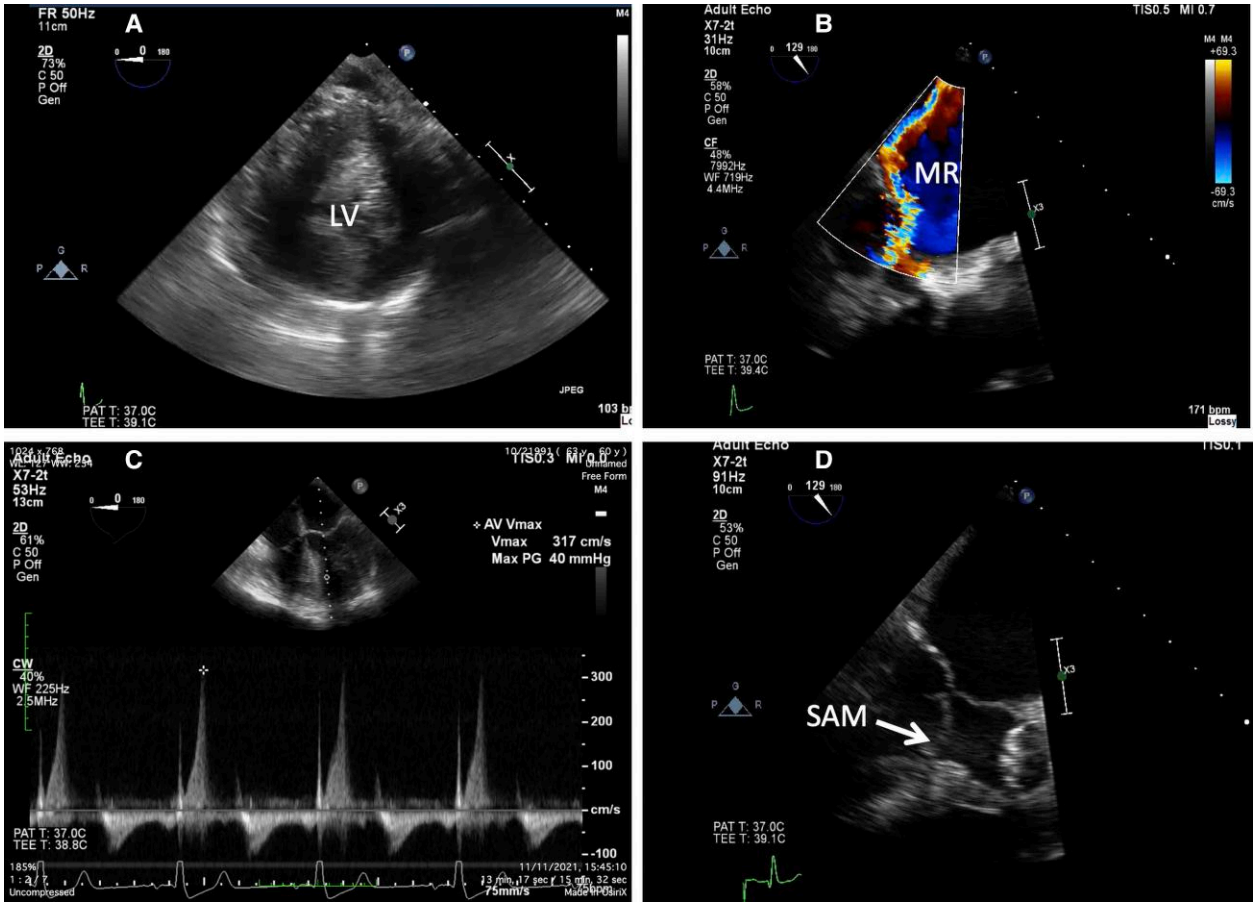

Figure 10 TOE in a patient after coronary artery bypass graft surgery who presented with worsening haemodynamics despite increasing inotopic support. (*A*) Transgastric short-axis view showing thickened LV with very small LV end-systolic volume. (*B*) Mid-oesophageal long-axis view at 120° showing eccentric anterior MR with colour Doppler. (*C*) CWD interrogation of LV mid-cavity denoting mid-ventricular peak systolic gradient 40 mmHg with late peaking indicating dynamic obstructive physiology. (*D*) Mid-oesophageal long-axis view at 120° showing SAM of the mitral valve (white arrow). The goal of the study was to assess haemodynamics and determine the cause of shock. The study showed that the patient has predominantly obstructive shock physiology due to dynamic LVOT and mid-LV cavity obstruction which requires changing management. This study requires advanced level of training in CUS. LV, left ventricle; MR, mitral regurgitation; SAM, systolic anterior motion of the mitral valve.

TTS (stress cardiomyopathy)

Among several clinical phenotypes of TTS, the most common is the apical ballooning, which is characterized by the akinesia of apex and/or mid-ventricular myocardial segments with a basal segments' hyperkinesia. The second commonest phenotype is the 'mid-ventricular', which is described as akinesis of the mid-segments with preserved or mildly reduced contractility of apical and basal segments.[116,117]

These common variants are characterized by a pattern of wall motion abnormalities which typically extends beyond the territory of a single coronary artery distribution.[118,119] Speckle tracking assessment may be useful even in the acute phase to confirm the 'circumferential pattern' and LV twisting abnormalities.[120] Other rare phenotypes exist (basal or focal phenotype) which may represent diagnostic challenge by CUS only and are usually confirmed only after exclusion of other possible aetiologies (i.e. CAD, myocarditis).

RV involvement is associated with worse clinical course, and it is usually defined by severe akinesis or dyskinesis, localized exclusively at the apical and/or mid RV segments (biventricular ballooning), with sparing of the basal segments ('reverse McConnell's sign').[121] Therefore, TAPSE in this case is not reliable, while RV free-wall longitudinal strain should be evaluated if facilities and expertise are available.

The most common complications of TTS include LVOT dynamic obstruction (12–25%)[122] with associated SAM of the mitral valve (SAM—15%)[123] and ventricular mural thrombi, especially in the apical ballooning form (1–2%).[124] Functional, transient, reversible mitral insufficiency can be associated with TTS with or without LVOTO (due to transient myocardial stunning involving the papillary muscles). Other complications could be inter-VSR or LV free-wall rupture.

Key messages:

- CUS allows the identification of Takotsubo phenotypes as well as potential complications.
- Myocardial strain imaging may be useful in evaluating the deformation pattern and other rare phenotypes of SCM.

Acute aortic syndromes

Acute aortic syndromes (AAS) represent a group of critical cardiovascular conditions, including acute aortic dissection, intramural haematoma and penetrating aortic ulcer.

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In suspected AASs, a systematic echocardiographic assessment should incorporate all available windows to visualize the thoracic aorta, including the right parasternal, suprasternal, apical long-axis (for the descending thoracic aorta), and subcostal views (for the abdominal aorta). Since TTE sensitivity in the quick rule in/out of acute ascending aortic dissection is 78–90%, but only 31–55% for descending aortic dissection,[125,126] TOE and contrast CT scan are usually warranted for a more detailed diagnosis and structural investigation. Integrating CUS with D-dimer has allowed the rapid triaging of high probability patients while safely and efficiently ruling out acute aortic dissection.[127]

Echocardiographic landmarks of AAS include:

(1) Identification of an intimal flap[128] or localized thickening (>5 mm), with or without an echolucent zone, as seen in cases of intramural haematoma, or the presence of intramural haematoma/aneurysm in penetrating ulcer scenarios.
(2) Assessment of aortic root valve and coronary sinus in case of Type A aortic dissection, and severity of AR, resulting either from dissection-induced dilation or valve leaflets involvement.[129]
(3) True and false lumen differentiation: CUS may distinguish between true and false lumens based on flow characteristics using colour Doppler, although during CPR this can be challenging. The true lumen typically appears smaller, shows laminar flow with uniform colour filling, and has a higher velocity on Doppler imaging. In contrast, the false lumen is often larger, demonstrates turbulent or sluggish flow, and may contain spontaneous contrast or thrombus. The intimal flap moves towards the false lumen in systole and towards the true lumen in diastole, reflecting pressure changes. Additionally, major aortic branches usually arise from the true lumen.[125]
(4) Extent and location of dissection: TOE can evaluate the extent of the dissection, including involvement of the ascending aorta, arch, and descending aorta.
(5) Pericardial effusion in acute Type A aortic dissection is a high-risk finding, indicating aortic rupture or impending rupture, and is associated with increased mortality due to cardiac tamponade and haemodynamic instability, necessitating urgent surgical intervention.

Key messages:

- In patients with chest pain, especially if associated with haemodynamic instability/collapse, the presence of (new) AR or pericardial effusion should raise suspicion of possible Type A acute aortic dissection.
- Aortic dissection cannot be ruled out based on TTE alone; therefore, TOE and CT are usually required.
- Care should be taken to avoid significant rise of blood pressure during TOE examination.

Cardiac arrest and peri-arrest situations

CUS offers a real-time insight into cardiac function during cardiac arrest, guiding resuscitation strategies during ALS protocol.[130]

Transthoracic CUS examination is typically performed first due to its non-invasive nature, and wide availability, whereas TOE may be employed for better visualization, particularly when transthoracic images are suboptimal, facilities are available, and patient is intubated. CUS is also helpful during extracorporeal CPR by providing real-time guidance which is essential for validation of support, guiding wire and cannulation, troubleshooting and weaning from mechanical circulatory support (MCS). However, the role of imaging in MCS is detailed elsewhere.[131,132]

Reversible (treatable) causes of cardiac arrest identifiable by CUS are: cardiac tamponade, severe hypovolaemia, PE, and myocardial infarction. Whereas the former applies also in the context of chest compression, the last three require return to spontaneous circulation for definitive diagnosis.

CUS must be performed during pulse check without interfering with chest compressions. Focused TOE (fTOE) provides continuous and high-quality images without the need to interrupt chest compressions,[133,134] suggesting underlined pathology (e.g. AAS, mechanical complications of AMI), monitoring LVOT VTI as sign of effective compression and guiding during eventual wiring and cannulation process for MCS.[135,136]

CUS examination after return of spontaneous circulation, and without profound hypothermia, allows a re-assessment of aetiological causes of cardiac arrest presenting with non-shockable rhythm.[137]

Key messages:

- Incorporating CUS into ALS protocol and the management of cardiac arrest enhances the ability to diagnose underlying pathophysiological conditions (especially treatable causes of cardiac arrest), tailor resuscitative efforts, and make informed prognostic decisions.
- CUS examination in cardiac arrest is challenging, and proper training and coordinated teamwork are essential for timely data acquisition and interpretation, while minimizing interruption of chest compressions during CPR.

Training and competence in CUS in cardiovascular emergency and critical care

Competence refers to the acknowledgement of one's capability or legal authority. An echocardiographer's competence in performing ultrasonographic examinations is determined by their knowledge, skills, and attitude. The European Society of Cardiology (ESC) core curriculum emphasizes two key aspects in imaging training: the importance of a multimodality approach and the redefinition of independence levels for performing and interpreting diagnostic techniques, including echocardiography. These new levels are described as 'Entrustable Professional Activities' (EPA).[138,139] These five levels start with Level 1, to observe; Level 2, direct supervision; Level 3, indirect supervision; Level 4, distant supervision; and Level 5, no supervision and able to teach.

Performing a CUS examination in cardiovascular emergency and critical care is challenging. Rapid decision-making is required in a stressful environment, often with difficulties in obtaining good quality images. The expansion of CUS beyond cardiology to a wider range of acute care specialties has led inevitably to variability in the level of practice and quality of scans. The gap between cardiology and intensive care medicine albeit becoming smaller yet it still requires ongoing work to train cardiologists in critical care pathophysiology and train intensivists in cardiovascular physiology and CUS. All these challenges can result to interpretation errors or missed critical abnormalities. CUS in cardiovascular emergency and critical care should be performed by anyone proficient in obtaining valuable information and utilising it in the decision-making process. This expertise includes the ability to obtain adequate images (imaging technique) and interpret them in the specific clinical context. Poorly acquired or low-quality images can result in inaccurate readings and potentially dangerous conclusions.[140]

In cardiovascular emergency and critical care, all CUS scans should be performed by competent and/or accredited practitioner or at least be supervised by an expert physician with advanced competence in critical care and emergency echocardiography (*Tables 5–7*).

Requirements for reaching competence in CUS in cardiovascular emergency and critical care

The type of CUS that is used in in cardiovascular emergency and critical care depends on the clinical problem, environment, machine availability, and competence of the operator. The more comprehensive the

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Table 5 Components of practical training required for practitioners performing advanced critical care echocardiography

- Indication and limitations of advanced critical care echocardiography
- Advanced knowledge about technical settings, artefacts, and pitfalls of echocardiography (PWD, CWD, tissue Doppler, strain imaging, contrast echocardiography, and 3D TTE and TOE)
- Advanced knowledge about anatomy of the heart and the great thoracic vessels
- Standard and advanced echocardiographic views
- Analysis of the thoracic aorta
  - Detection of acute aortic dissection (haematoma, ulcer)
  - Detection of ectasia and aneurysm
- Detection of PV malfunction
- Detection and quantification of acute valvular diseases
- Assessment of pericardium
- Assessment of cardiomyopathies
- Detection and differential diagnosis of cardiac masses—cardiac sources of embolism
- Analysis of left and right cardiac chamber size and function
  - Assessment of cardiac output, detection of CS and its differentiation from other causes of shock
  - Detection of regional left ventricular dysfunction
  - Assessment of pulmonary vascular resistance
  - Assessment of pulmonary artery pressures
  - Assessment of LV and RV diastology
- Assessment of systemic venous congestion
- Assessment of preload responsiveness
- Assessment of pulmonary consolidation

3D, three-dimensional; TTE, transthoracic echocardiography; TOE, transoesophageal echocardiography; LV, left ventricle; RV, right ventricle.

Table 6 Proposed list of CVDs/conditions to be included in additional theoretical learning programme for non-cardiologists performing emergency and critical care echocardiography[25])

- Acute coronary syndrome/AMI
- Mechanical complications of AMI
- AAS/aortic dissection
- Acute PE
- Acute HF/CS
- Acute pericarditis
- Cardiac tamponade
- Acute myocarditis
- Pneumothorax
- Cardiomyopathies
- Aortic stenosis
- Acute valvular regurgitation
- Hypertrophic cardiomyopathy
- Takotsubo cardiomyopathy
- PV dysfunction
- Cardiac sources of embolism (tumours and masses)
- Ventricular assist device malfunction
- Acute complications of interventional procedures in the catheterization and electrophysiological laboratories
- Acute complications of cardiac surgery
- Endocarditis
- Traumatic injuries of the heart

education and the larger the experience of the individual performing CUS, the higher will be the diagnostic accuracy and, consequently, the better patient management. CUS examination can be performed by any general cardiologist, intensivist, anaesthetist, or acute care physician who is trained to obtain necessary information by ultrasound, to interpret it adequately and to efficiently use it actual clinical context.

Medical professionals should be aware of the existing gap between adequate competence and experience needed to properly address both simple and complex issues in the emergency setting, and the trend towards the use of CUS by non-cardiologists and cardiologists with insufficient formal training. Therefore, it should be emphasized that CUS in the emergency setting is a highly demanding procedure and, due to serious implications of the examination result, should not be attempted by inexperienced operators without supervision.[25]

Even for trained cardiologists, to achieve basic competence in emergency and critical care CUS, it is strongly recommended that they complete additional training, including the interpretation and reporting of at least 150 CUS examinations in emergency cardiovascular and/or critical care scenarios, to enhance their technical skills and experience in emergency and critical care CUS (*Table 7*).[6]

For non-cardiologists, additional education and training are required to act as basic-level operators in emergency and critical care CUS.[25] This training should encompass specific theoretical knowledge of emergency cardiovascular conditions (*Table 6*) and practical hands-on training to develop the technical skills necessary for performing adequate echocardiographic studies in challenging emergency and critical care settings (*Table 7*). To attain basic-level operator proficiency, non-cardiologists should interpret and report and 150 emergency and critical care CUS cases, covering a full range of emergency cardiovascular conditions, with at least 50 of these cases performed, documented, and interpreted under close supervision (*Table 7*).[25] This recommended basic-level training corresponds to EPA Level 5 competence in general adult TTE, EPA Level 3 competence in TOE, and EPA Level 2 in SE.

'Advanced critical care CUS', on the other hand, is often even more complex and typically requires an advanced level of echocardiography training as well as an understanding of complex haemodynamic alterations and heart-lung interactions (*Table 5*). This recommended advanced-level training corresponds to EPA Level 5 competence in general adult TTE, EPA Level 5 competence in TOE, and EPA Level 5 competence in SE.

It is crucial that all programme directors involved in the education and training of CUS in cardiovascular emergency and critical care possess advanced expertise in echocardiography, having achieved Level 3 competence in general adult TTE and TOE as per the ESC core curriculum.[141] Sonographers and fellows in training should not routinely perform these exams unsupervised, and only in life-threatening scenarios, where any delay could be fatal, may they operate independently. These examinations must be re-interpreted by experts as soon as possible. Establishing optimal logistics for the emergency echocardiography service (as discussed below) should minimize these situations.[25] Continuous supervision is essential for the success of such a service.

Detailed proposals for reaching competence in FoCUS can be found in the recently published EACVI document on FoCUS core curriculum and core syllabus.[142]

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Table 7 Minimal requirements for training and expertise for cardiologists and non-cardiologists for independently performing/interpreting CUS in cardiovascular emergency settings (modified from Popescu *et al.*, Neskovic *et al.*)

Level of competence	Profile of individual performing emergency and critical care echocardiography	Minimal number of examinations performed to become competent	Achieved level of expertise according to EAE recommendations	Level of competence to be achieved according to ESC Core Curriculum	Additional education/ training requirements

Basic-level operator in emergency and critical care CUS)	'Cardiologists' who completed training according to ESC Core Curriculum requirements for general training for cardiologists) and 'non-cardiologists' completed training in their own specialties	350 (TTE)	Basic echocardiography	• Level 3 in general adult TTE
				• Level 2 in TOE
				• Level 1 in SE
					• Mandatory for non-cardiologists
					  • Theoretical: specific knowledge on emergency CVDs/ conditions (see list, *Table 8*).
					• Mandatory for all:
					  • Practical skills: 150 emergency cases interpreted/ reported; 50 of which performed and reported under close supervision.

Advanced-level operator in Emergency and critical care CUS)	Both cardiologists and non-cardiologists (e.g. anaesthesiologists, intensivists) (completed training in their own speciality with subspecialty interest in echocardiography)	750 (TTE) 75 (TOE) 100 (stress echo)	Advanced echocardiography	• Level 3 in general adult TTE • Level 3 in complex adult TTE • Level 3 in TOE • Level 3 in SE	Advanced-level operators may opt to pursue further specialist training in ECMO and MCS

Proposed levels of competence for CUS in cardiovascular emergency and critical care

We propose two levels of competence in emergency and critical care echocardiography: the independent basic operator level and the advanced operator level (*Table 7*).[25]

'Independent (basic) operator level is achieved by both cardiologists and non-cardiologists who successfully completed education and training according to the proposed recommendations (*Table 7*).

'The advanced operator level' is reserved for practitioners who have completed training equivalent to the advanced echocardiography level of expertise as defined by the EACVI recommendations.[138] This includes specific training and competence in special echocardiographic procedures, such as TOE and contrast echocardiography. Advanced-level operators should be individually certified in echocardiography at the highest national or international level. They should be responsible for education, training, supervision, and quality control, and act as key figures in establishing institutional emergency and critical care echocardiographic services.[25]

Completion of an appropriate training programme does not necessarily ensure competence. Competence should be formally assessed through a certification process. Currently, the EACVI has not established such a process for emergency and critical care echocardiography, but individual certifications for various echocardiographic modalities and laboratory accreditations are offered. Considering the proposed requirements for an independent basic operator level in emergency echocardiography (*Table 6*), individual certification can be achieved through the standard EACVI certification process for TTE. Additionally, for the advanced operator level, individual national/EACVI certification in TOE is advised. Currently, there is ongoing preparation of the EACVI certification process for FoCUS.

As individual competence, especially in emergency medicine, is strongly related to the competence of the team and facilities around them, successful EACVI laboratory accreditation is likely to ensure a favourable environment for providing high-standard service in all echo modalities, including emergency and critical care echocardiography.

Maintenance of competence in emergency and critical care echocardiography

Ongoing practice and continuous learning are essential to maintain competence in emergency and critical care echocardiography over time. The EACVI has established basic requirements for re-certification in TTE and TOE, which include ongoing practice and proof of continuing education, such as participation in relevant scientific meetings (e.g. certificates of attendance).[138] In health systems where physicians primarily report studies performed by sonographers, it may be necessary to distinguish between the number of studies reported and those personally performed to ensure ongoing competence.

For emergency echocardiography, exposure to 50 emergency cases per year (discussed, performed, and/or interpreted) with a diverse case

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Table 8 (adapted from Neskovic et al.) 'ABCDE' approach

A	Awareness	• Fight against routine • Think beyond apparent explanations
B	Be suspicious	• Referral diagnosis may be misleading • Never trust, confirm • Be aware of echocardiographic pitfalls
C	Comprehensiveness	• Do as complete examination as suitable • Careful interpretation
D	Double R (record and review)	• The study should be recorded and reviewed • Teamwork is crucial
E	Education and research	• Reflection and teaching • Audit • Clinical governance • Research

This approach summarizes mental roadmap which should be followed in order to handle real-life challenges which may lead to diagnostic and interpretation errors.

mix is considered reasonably sufficient to maintain competence for both cardiologists and qualified non-cardiologists. For advanced critical care echocardiography, we propose that exposure to[93–118] echocardiograms in critically ill patients per year (discussed, performed, and/or interpreted) with a diverse case mix is considered reasonably sufficient to maintain competence for both cardiologists and qualified non-cardiologists.[25]

Governance and medicolegal aspects

In order to reduce diagnostic errors, the 'ABCDE approach' is proposed (*Table 8*, adapted from Neskovic *et al.*[142]

Performing and reporting of emergency and critical care CUS

'ABCDE' approach

Table 8 (adapted from Neskovic *et al.*) summarizes 'ABCDE' approach. This approach summarizes a mental roadmap which should be followed in order to handle real-life challenges which may lead to diagnostic and interpretation errors.

Study report of emergency and critical care CUS

The emergency and critical care CUS study report should adhere to the basic format recommended by the EACVI.[144] However, given the often-limited time available for report generation, an initial preliminary report focusing on critical findings and integrated into the decision-making process may be issued. The critical care CUS report should include the following: patient's haemodynamic data at the time of scanning, inotropic/vasopressor support and dosing, ventilatory settings and any other haemodynamic support (e.g. MCS). The interpretation of CUS data should always be made in the context of catecholamine therapy and mechanical ventilatory support. If the study results indicate the need for urgent treatment, the physician responsible for the patient must be informed directly.

Medicolegal aspect

Given the higher likelihood of diagnostic errors in emergency and critical care settings, medical professionals must be fully aware of the potential medicolegal consequences, and this should be in the context of a fully governanced system. It is crucial that all emergency cases are conducted using appropriate ultrasound machines and that the images and data obtained are well-documented and archived safely. Stored images and clips can be used later for case reviews and serve as evidence of the findings in the acute setting. Reports should always reflect the recorded findings that have been interpreted, approved, and signed by individuals with adequate training and accreditation. Training scans must not be stored in the hospital archiving system unless they are reviewed and approved by an accredited practitioner. Images and clips obtained with HUDs should also be stored, and the results (not a formal report) should be written in the patient's file only after they are reviewed, approved and signed by practitioners with adequate training and accreditation.

Efforts should be made to obtain informed consent for potentially hazardous procedures (e.g. TOE) from the patient or their family, whenever applicable, although this may not always be possible in life-threatening situations.

Handheld ultrasound devices

A range of miniaturized, pocket-size HUDs are available on the market, offering a good quality 2D, colour Doppler and, to some extent, PWD, CWD and even TDI, which represents a huge technological advancement as well as a challenge for ensuring these tools are utilized safely and after appropriate training. A broad range of medical practitioners are currently using these devices, typically to enhance bedside physical examinations since they are generally sufficient for rapid initial screening in an emergency setting and to complement a physical examination in CCU and ICU settings.[145] They allow for qualitative evaluation of global LV and RV function, detection of pericardial and/or pleural effusion, identification of B-lines in the lungs (indicating extravascular lung water), assessment of the size and respiratory changes of the IVC, and identification of gross valvular abnormalities. The increasing availability of PWD in today's HUD as well as artificial intelligence (AI) algorithms provides an opportunity for more advanced assessment. AI-enabled devices are now able to provide a comprehensive CUS report based on a few basic echocardiographic views obtained by the operator.[146,147]

Nevertheless, examinations with current HUD are more suited for FoCUS or targeted assessments at the point of care, and they still cannot replace assessment by cart-based echocardiography machines for complex CUS studies. Recent observational data showed that HUD delivered results comparable to TTE in patient presenting to the emergency department with ACS, offering a faster and more efficient imaging alternative that provides adequate information for clinical decision-making in most cases.[148] Routine CUS assessment significantly influenced management in half of the patients, highlighting its clinical value in this population.[148]

Future perspectives

Future perspectives on emergency and critical care CUS are poised to be significantly transformed by advancements in AI, big data, and machine learning. AI algorithms have the potential to enhance diagnostic accuracy by rapidly analysing large volumes of echocardiographic data, identifying subtle patterns and anomalies that might be missed by the human eye.[149] For instance, AI can assist in the automated detection of cardiac

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pathologies, such as valvular heart diseases and cardiomyopathies, through real-time image analysis, and can even provide complete echocardiogram interpretation with high accuracy.[150] Thus, machine learning models, which can continuously improve by learning from vast datasets, might enable more precise and personalized patient assessments and generate accurate CUS reports. In addition, these models may potentially predict patient outcomes, assist in risk stratification, and support decision-making processes in emergency cardiovascular and critical care settings.

The integration of big data allows for the aggregation and analysis of diverse patient information, facilitating comprehensive decision-making and predictive analytics. Big data analytics can uncover trends and correlations across large populations, providing insights that can enhance patient management and treatment strategies.[151] For example, the combination of echocardiographic data with other clinical data may help in developing predictive models for patient deterioration, allowing for timely interventions.

AI and machine learning also may play a crucial role in facilitating critical care and emergency echocardiography education by providing 'interactive training modules and simulation platforms' that help trainees develop their skills more effectively. These technologies can offer personalized feedback and adaptive learning pathways, enhancing the learning experience and proficiency of echocardiography students.

Together, these technologies promise to revolutionize emergency and critical care CUS by expediting assessment, increasing efficiency, enabling real-time, data-driven clinical interventions, and improving patient outcomes. The use of AI and machine learning might reduce the cognitive load on clinicians, allowing them to focus more on patient care rather than data interpretation. Moreover, these advancements can democratize access to high-quality echocardiographic interpretation, making it possible for less experienced practitioners to provide accurate assessments.

The Internet of Things (IoT) in hospitals further enhances these capabilities by enabling seamless integration of echocardiographic data with other patient monitoring systems, facilitating continuous and comprehensive care. These IoT connected systems can improve decision-making and patient safety through automated alerts, trend analysis, and remote monitoring capabilities.[152]

Despite their promise, these technologies present several challenges. Overreliance may lead clinicians to lose critical interpretative skills. Concerns around data privacy, cybersecurity, and unequal access—especially in low-resource settings—remain significant. Moreover, the high costs of deployment and upkeep may hinder broader implementation, limiting their integration into routine clinical practice.

As these technologies continue to evolve, they will undoubtedly play an integral role in shaping the future of emergency and critical care CUS. Despite their challenges, they hold immense potential to transform acute cardiac care, paving the way for a new paradigm in delivering high-quality, efficient, and equitable patient care.

Conclusion

CUS is an essential tool in cardiovascular emergency and critical care, enabling rapid diagnosis, risk stratification, and management of critically ill patients. This clinical consensus statement highlights its key applications, including the pathophysiological basis of the assessment of various clinical conditions.

Standardized training, certification, and structured competency levels are crucial to maintaining high-quality imaging and accurate interpretation in acute settings. By establishing practical bedside guidance, this document provides a framework for optimising the use of CUS in emergency and critical care settings. Its widespread adoption will contribute to improved patient outcomes by facilitating earlier detection, targeted interventions, and more effective haemodynamic monitoring in high-risk cardiovascular conditions.

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Data availability

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